THE EFFECTS OF VISUAL ACTIVITIES AND PARENTAL HISTORY OF MYOPIA ON OCULAR DEVELOPMENT AND REFRACTION IN PRE-SCHOOL CHILDREN

BY

MAKSUDUL ISLAM

DEPT OF OPHTHALMOLOGY AND VISUAL SCIENCES
THE CHINESE UNIVERSITY OF HONG KONG

THESIS
SUBMITTED TO THE GRADUATE SCHOOL OF
THE CHINESE UNIVERSITY OF HONG KONG
IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

MASTER OF PHILOSOPHY IN OPHTHALMOLOGY

HONG KONG
DECEMBER, 1995

LIST OF ABBREVIATION

AL :	Axial length
ACD :	Anterior chamber depth
CI :	Confidence Interval
CC:	Corneal curvature
cm :	Centimetre
cyl :	Cylinder
D :	Diopter
& :	And
H :	Home
hr :	Hour
IOP :	Intraocular Pressure
LT :	Lens thickness
LE :	Left eye
LP:	Lens power
min :	Minute
mm :	Millimetre
mm Hg :	Millimetre of mercury
mth :	Month
N :	Number
NA:	Not available
PH :	Pin hole
RE :	Right eye
S :	School
SPEQ :	Spherical equivalent
SPH :	Spherical
SD :	Standard deviation
SE :	Standard error
VL :	Vitreous length
wk :	Week
yr. :	year

ACKNOWLEDGEMENTS

I would like to express my deepest gratitude and sincere thanks to my supervisor Dr Dennis S C Lam and Chairman of the department Professor Mark O M Tso for their constant advice, guidance with patience, support and encouragement all through my research.

I wish to thank Dr Winnie Li & Miss Eva Cheung for interpreter and other help during my investigation period. I also extend my thanks to Dr Dorothy Fan for checking datas and statistical analyses. I should thank Dr John Michon for his helpful comments on the manuscript on my thesis writing.

I am also greatly indebted to the Myopia International Research Foundation, Inc. (USA) for providing me with the latest information.

I also expressed my thanks to all teachers, doctors and friends of local and overseas for their intimate co-operation and encouragement.

My appreciation also goes to my family, Government of Bangladesh, children and their parents and teachers of Liu Yan Tak Memorial Kindergarten, Li Ka Shing Eye Specialist Clinic, Li Ping Medical Library, CCTER for statistical analyses, Department of Ophthalmology and Visual Sciences, Division of Surgical Sciences, Faculty of Medicine and the Chinese University of Hong Kong for their help.

CONTENTS

CHAPTER 1

ABSTRACT

Purpose: This study was done to assess the relationship between parental history of myopia and the performance of visual tasks on the development of myopia in pre-school children in Hong Kong. **Methods:** After meeting inclusion criteria and providing informed consent, an activity diary measuring visual tasks was recorded in school and at home in 237 subjects aged 36-83 months (mean 59.43 months). In addition, detailed personal histories and any parental history of myopia were recorded. Subjects underwent cycloplegic refraction and ultrasound biometry. Refractive errors greater than -0.50 D were regarded as myopic and the spherical equivalent was calculated. **Results:** The prevalence of myopia in the children was 4.22%, with values for emmetropia of 28.69% and for hyperopia of 67.09%. The mean spherical equivalent and axial length of the children in which neither parent was myopic (n=120) were +0.93 D and 21.98 mm. If either parent was myopic (n=78) the mean values were +0.77 D and 22.01 mm. If both parents were myopic (n=39) the mean values were +0.82 D and 21.86 mm. No statistically significant correlation was found between parental myopia and refractive error and axial length in their children in this study group ($p > 0.05$ & Wilks Lambda: 0.961 for all models). In this population the visual tasks were measured as diopter-hours per week. This was calculated as reported working distance and amount of time spent (time x 1/distance) on various visual tasks. Although duration of visual activities increased from 3 to 6 years, there was no significant correlation of visual task time with refraction and axial length (Wilks Lambda: 0.959 for all models). There was a relationship between increased axial and

vitreous length with increased age and myopic refractive error of the children. Myopia usually correlates well with axial and vitreous lengths, however these results suggest that age and increased axial and vitreous length have more influence on the development of myopia. **Conclusion:** These data suggest that parental myopia and the degree of visual effort were not important predictors of eye size and refractive status in pre-school children in Hong Kong. There was a relationship between age and refraction with axial and vitreous length in these children. No relation was found between parental myopia and education and their children's refractive status. The risk factors for myopia in pre-school children are still controversial. Much more work must be done to identify the major factors which cause myopia, and their relative importance still remain to be elucidated. A long-term follow-up study to assess these factors may be necessary.

CHAPTER 2

2.1 INTRODUCTION

Myopia (short-sightedness) exists when parallel rays of incident light focus in front of the retina when the eyes are not accomodating.[1] Clinically, myopia may be defined as a mismatch in the refracting optics of the eye and its length, causing images to be focused in front of the retina (Zadnik et al., 1994).

In this situation distant objects cannot be seen clearly. Myopic eyes have a finite far point in space from which divergent rays of light are brought to a focus on the retina without accommodative effort.[2] The word myopia comes from the Greek word "myein" meaning "shutting" the eye, to characterize those individuals who squint and narrow their eyelids to improve distance visual acuity with a pinhole effect.[3,4] Myopes may habitually try to narrow their palpebral fissure to form a stenopaeic slit to improve the image quality.[5] Myopia can arise because either the primary refractive components (cornea and lens) are too powerful or the globe is too long.[3] Lower degrees of myopia usually occur when the surfaces of the cornea and lens do not neutralize the effect of increasing axial length during growth (Sorsby and colleagues, 1957).

The prevalence of myopia in the normal full term new-born is between 4% and 6% (Curtin, 1985). The incidence of myopia remains fairly stable over the first few years of life but shows an increase with advancing age through childhood. This increase is seen when children are beginning school and this observation has implicated visual tasks and accommodation in the development of myopia.

The commonest form of myopia is juvenile-onset or school myopia which typically begins between the ages of 8 and 14 years. The development of refractive errors in school children and particularly the incidence of myopia has

been the object of extensive study since the last century. Neonates are usually hyperopic[4] and gradually approach emmetropia. Older children and young adult are tend towards myopia while middle-aged persons are less myopic.[7] Myopic neonates are four times more likely than hyperopic neonates to exhibit myopia later in life (Banks, 1980). A high degree of congenital myopia may occur occasionally (Harman, 1913, 1914; Jackson, 1932; Hofmann and Carey 1942). The majority of cases of myopia first appear between the age of five and puberty and may progress as growth continues until the refractive status stabilizes after adolescence is passed.[7] Most investigators have found that a gradual reduction in hyperopia occurs during later infancy and childhood (Hirsch, 1963; Millodot, 1972). This may be caused by one or a combination of changes in the optical components of the eye including the cornea, anterior chamber depth, thickness of the lens, vitreous length and axial length.[8,9] Increased corneal or lenticular curvature or an increase in the lens index of refraction may cause myopia but more commonly myopia is the result of axial elongation of the posterior segment of the eye.[3] Since the axial length of eye increases gradually with age, one would expect the apparent hyperopic error to shrink concurrently. Millodot (1972) has suggested that the average child may be nearly emmetropic from infancy through adolescence. Because the optical components grow from infancy to puberty, the eye's growth must be a coordinated process to maintain emmetropia. When accommodation is increased, ciliary muscle activity facilitates axial growth of the globe, whereas when accommodation is relaxed, axial growth is impeded (Young, 1977; Greene 1980; Ebenholtz, 1981 & Raviola et al., 1985). Thus, a young myopic eye, with little demand for an increase in accommodation, would become less myopic with age due to the hypothesized impediment of axial growth.

Animal studies (Raviola & Wiesel, 1985) and clinical observations have noted that anomalous visual experience may trigger the development of myopia in the immature visual system. Although myopia had been widely studied for over 100 years, the relative contribution of genetic and environmental factors in

its etiology have not been clarified.[12] Myopia in humans may be caused by a number of different structural abnormalities, such as elongation of the eye or changes in the curvatures of its refracting surfaces.[30] Hence, the detection and correction of refractive error in children is important for two reasons (Tongue AC, 1987): a) to prevent irreversible vision loss secondary to amblyopia and b) to treat visual impairment detrimental to the child's normal functioning in his or her daily life.[47] Myopia in children appears to result from the cumulative effects of axial elongation and an inadequate compensatory decrease in lens thickness, curvature and possibly refractive index (Zadnik, Mutti, Friedman, Sholtz & Adams, 1994). Earlier studies have shown that these refractive components undergo the most significant and rapid changes during the first 3 years life, while excessive or anomalous axial elongation is associated with the second stage of ocular growth, a slower developmental phase lasting from ages 3-15 years (Sorsby, Benjamin & Sheridan, 1961). Subsequent reports have concluded that the majority of changes in anterior and posterior chamber lengths are completed by the first or second years of life, respectively (Larsen, 1971).

Two general theories may be considered, namely genetic and environmental or "use-abuse". Hereditary theory predicts that the differences in refractive error among ethnic groups results from a genetic cause and that the increase of incidence rate of myopia among school children is related not to school experiences but to age. In contrast, those who advocate the "use-abuse" theory believe that school experience is a significant factor in causing myopia.[58] Several genetic studies have demonstrated a significant familial influence in myopia. The environmental theory hypothesizes increased scleral stress by the extraocular and intraocular muscles during convergence and increased ciliary muscle tone during accommodation. In any case, a normal increase in axial length usually offsets mild hyperopia in young children. This growth pattern has been well-described and has been termed emmetropization (Sorsby et al., 1961; Mohindra & Held, 1981; Gwiazda, Thorn, Bauer & Held,

1993). Continuing axial elongation without a compensatory reduction of lens power, however, may result in myopia.[53]

The prevalence of myopia varies with age and geography. In the USA the prevalence of myopia was measured at 29.3% at the age of 12 and 33.2% at the age of 17.[21] In Taiwan the prevalence of myopia among school children increased from 4% at the age 6 years to 40% at the age of 12 years. At the age 15 the myopia prevalence was over 70%.[59] Another Taiwanese study showed only 2% of eyes were myopic up to 6 years of age. The same group was followed-up after 4 years and myopic rate had increased to 27.3%. The prevalence was as high as 92.9% among the university students.[37] A high prevalence of myopia was found among Chinese, Japanese and Koreans. In contrast a lower prevalence of myopia was found among Indians[11] affecting 25% of the population and up to 80% of school children. A combination of heredity and environmental factors may be the reason for the high prevalence rate of myopia in Chinese.[92]

It is difficult to compare studies because of different inclusion criteria and definitions of myopia among different authors. Lam and Goh studied 383 school children in Hong Kong and measured a mean refraction of -0.09 D at the age 6 to 7 years, -1.31 D at the age of 12-13 and -2.05 D at the age of 16 to 17 years. The prevalence of myopia in these age groups was 31.4%, 58.1% and 58.8% respectively.[50] Myopia starts at an earlier age and that it progresses more quickly among Hong Kong Chinese than in non-Chinese populations (Edwards M, 1991). Previous studies have shown that those who spend more time in educational efforts have a higher myopia prevalence than those spending less time in education. It has also been stated that children may have longer eyes in myopic families(Zadnik et al, 1994; Teikari et al, 1991).

CHAPTER 3

3.1 OBJECTIVES

- To assess the influence of visual tasks on the development of myopia in a group of Hong Kong pre-school children. The child's visual task was measured by calculating the diopteric distance of visual work and the time spent in each type of visual activity. A week-long diary of visual activities (hours of reading and writing, watching television etc.) of each child was obtained through the participation of school teachers and parents.
- To study the influence of parental myopia and educational level on these children. A questionnaire on parental history of myopia and educational level was administered for every subject and summarized through multivariate analysis.
- To study the distribution of refraction of myopia among pre-school children in Hong Kong. Each child underwent a complete eye examination including cycloplegic refraction with auto-keratorefractometer and axial and vitreous length of the eye globe by ultrasonic biometry.

CHAPTER 4

4.1 LITERATURE REVIEW

4.1.1 BACKGROUND

The term myopia originates from a Greek word which means contracting or closing the eyes. It describes the typical facial appearance of a myope as he or she attempts to obtain clear distance vision. Until the introduction of spectacles, squinting the lids with the resultant production of a horizontal slit was the only practical means for obtaining clear distance vision (Curtin, 1985). Aspects of human myopia have been studied for at least 80 years (Stieger, 1913; Working Group on Myopia Prevalence and Progression, 1989). This research has produced evidence of a genetic etiology for myopia as well as evidence to support an environmental theory of myopia centered on accommodation and near work (McBrien & Barnes, 1989). Despite intensive study, the precise etiology of myopia remains unknown. The clinical literature is replete with genetic, environmental and dystrophic theories of myopia (Curtin, 1985; Tokoro et al, 1967; Greene, 1980; Gross, 1982, Jensen, 1991). It is as yet unknown what relative roles these "nature" and "nurture" components play in the onset and progression of human myopia, and there are many limitations in attempting to discern their relative roles through epidemiological and clinical research. In 1913 Steiger asserted that the concept of axial myopia could not explain all cases and found that corneal refraction ranged from 38 to 48 D. He postulated that corneal curvature and axial length were independent variables. His theory refuted the idea that axial length is the sole cause of myopia and it brought to attention to the possibility of other variables in the eye itself. It also emphasized the importance of

hereditary rather than environmental influences. For the first time myopia was considered to be a physiological variable depending upon corneal curvature and axial length each of which possessed a normal range of variation. Steiger (1913) described emmetropia and ametropia as points on a normal distribution curve, with corneal power and axial length as independent variables. These concepts brought an entirely new approach to the study of myopia. Berg, Sorsby and co-workers found that an emmetropization effect was noted in distribution curves of refraction as a result of a correlation of corneal power and axial length. In ametropias of 4 D and above, this correlation appeared to break down. Their study also indicated that neither the lens nor the anterior chamber depth was an effective emmetropization factor. Tokoro and Araki (1967) asserted a high correlation of total refraction with axial length. This is true especially in the emmetropic range of refraction in which the correlation of corneal power diminishes the impact of axial length upon the refraction. Axial length remains for the most part the primary determinant of refractive status. Clinically significant myopia usually becomes manifest in late childhood, increasing slowly in degree for some years thereafter (Duke-Elder, 1970).[22] A history of either parent having myopia had the highest test specificity (probability of hyperopia more than +0.50 D at school entry given no juvenile myopia = 0.91).[55] The biological theory of myopia views myopia as the result of genetically determined characteristics of eye tissues, whereas the use-abuse theory views myopia as the result of habitual use of the eye at a near focal length. Myopia varies over age, gender, race, ethnicity, level of education, social class and degree of urbanization (Curtin B J, 1985; Angel et al. 1980).

4.1.2 GENETIC AND ENVIRONMENTAL STUDY

There may be a relationship between parental history of myopia and near work on the development of myopia in pre-school children. The strength of the correlation between parental history of myopia and axial length might

vary with the level of diopter-hours. A dose-response relationship may exist with the number of myopic parents.[15] Even before the onset of juvenile myopia, children of myopic parents may have longer eyes (Zadnik, 1994). Human myopia may be influenced by genetic factors because children with two myopic parents are slightly more likely to be myopic and have longer eyes than children with no myopic parents.[17] The premyopic eye in children with a family history of myopia already resembles the elongated eye present in myopia.[17] The growth pattern that normally keeps normal eyes emmetropic may be derailed in the myopic eye. This derailment becomes more likely as the eye enlarges. Therefore the larger an eye initially, the greater the risk of developing myopia.[15]

Normal ocular development generally proceeds towards a hyperopic state in which the axial length may be shorter than the focal length. As normal development progresses toward emmetropia, the axial length matches with the normal growth of the eye.[16] Juvenile-onset myopia usually develops between the ages of 8 and 14 years. Although less than 2% of children beginning kindergarten are myopic, more than 15 % of children are by the time they complete grade school at age 14 or 15 years.[18] A greater prevalence of myopia exists among the children of myopic parents than among the children of nonmyopic parents (Gwiazda, Thorn, Bauer & Held, 1993; Goldschmidt, 1968; Aston, 1985). The children with both myopic parents are on average less hyperopic and have deeper anterior chambers, longer vitreous chambers compared with children with no myopic parents.[18] Zadnik et al. found that 12.2% of the children with two myopic parents were myopic, 8.2% of the children with one myopic parent were myopic, but only 2.7% with no myopic parents were myopic. Overall, 7.5% were myopic. Work on emmetropization has described the relationship of refractive change to ocular growth, but has not yet elucidated the mechanisms responsible for the refractive change (Van Alphen, 1961; Sorsby et al., 1957; Hofstetter, 1969; Mark, 1972; Carroll, 1982).[29] The susceptibility to myopia is maximal early in life and declines

thereafter.[29] It has been observed that the prevalence rate of myopia on their parents in the high myopic group (71.0%) was significantly higher than that in the group of nearly emmetropes (33.3%). There was a slight correlation with their previous school performance, daily amount of near visual display work and preferential working distance.[31] In studies done in India to assess the interaction of environment and genetics, it has been postulated that myopes without family history of myopia had the highest amount of near work.[100]

It is generally accepted that hereditary plays a important role in refraction. In studies of monozygotic and dizygotic twins, monozygotic twins were much more likely to have similar refractive errors than dizygotic twins (Goldschmidt, 1968; Karlsoon, 1974). Several studies of the inheritance of refractive errors have demonstrated a significant family prevalence of myopia.

According to the environmental theory, increased scleral stress by the extraocular muscles during convergence and increased ciliary tone occurring during accommodation. This is the classic association of the onset of myopia with schooling (Curtin, 1985). However, no conclusive evidence proved that the near work was a cause of myopia. For example, increased intraocular pressure may cause the expansion of the globe. Whether environmental factors produce weakness of posterior sclera leading to increased axial length and myopia is still unknown.

The pathogenesis of the rising incidence of myopia is still obscure and is probably multifactorial. Only 5% to 8% of myopia is believed to be hereditary, so environmental factors must play a role in the development of myopia in young children.[34] These results suggests that it is less likely that the children of non-myopic parents will become myopic compared with children with one myopic parent. Myopia risk is highest when both parents are myopic.[67] When there is no parental myopia, 7.3% of the 7-year old children are myopic. An autosomal recessive model predicts 6.25% of expected myopia and when one parent is myopic, 26.2% of the 7-year-old children are myopic and in this group autosomal recessive model predicts 25% of expected myopia.[67] It has been

found in the Chinese population that myopia may be coupled with a high education level, and that the effect of environmental and cultural factors are more important than hereditary influence in the pathogenesis of myopia.[83] Twin studies among the Taiwanese students showed that there were significant differences in axial length between the monozygotic and dizygotic, ocular refraction was increased in monozygotic twins, but variation of ocular refraction with age and myopic progression correlated that the environmental factors might affect the eye in the way of myopization, so both the factors could interact in the development of myopia.[99] Studies in Chinese twins (C J Chen et al, 1985) for the conventional comparison of intrapair concordance between monozygotic and dizygotic twins, and a significant genetic influence was observed in addition to environmental factor and vice versa. A North American study on the refractive findings in 10 pairs of twins found a correlation of +0.99 between identical twins and +0.67 in fraternal twins (Wixon, 1958). This suggests that the closer the genetic tie, the greater the similarity in refraction. This was supported by a British study which found that ocular components and refraction were more closely correlated in identical twins than fraternal twins (Chen C J et al, 1988; Diamond et al, 1985 & Hirsch et al, 1969). The Newfoundland population study (Bear et al, 1981) provided evidence that familial similarities in education and near work increase resemblance in refraction among first degree relatives. A random sample of 1200 twins in one age stratum of the cohort was studied through questionnaire. The difference of refraction between two eyes were not significant. The mean difference in refraction between the monozygotic twins was significantly lower than that between dizygotic twins. This result suggest that inheritance has a substantial effect on the etiology of myopia.[101] At the same time environmental influences operate to bring about changes in refractive characteristics. Monkey studies by Young et al (1961, 1963, 1965) have demonstrated the effects of restricted visual space on the growth and refractive characteristics of the human primate

eye and indicate that environmental stress is capable of developing true axial myopia in the subhuman primate eye.[102]

Children in grades 1 through 4 showed a frequency of 10%; in grades 5 through 8, 14%; and in grades 9 through 12, 16%. Students in high school 10% involvement and those in college 28.5%. It was found that 1.4% among children ages 6 to 7 years to 9.1% in those 12 years and above were myopic.[2] Earlier studies by Sorsby (1933) found the mean refraction at age 3 years to be +2.65 (D). Brown (1938) & Slataper (1950) in their study observed that an increase in mean hyperopic refraction was recorded up through the seventh year. During the period of a year and a half Cohn (1886) found that 17% of 84 children who refracted from 0.00 D to + 1.00 D became myopic. In a group of 54 myopic schoolmates, 41% became more myopic. Sperduto and co-workers (1983) concluded that accommodative effort during the development of the eye is capable of producing axial elongation. Other studies found that there is an association between high hyperopia and subnormal intelligence (Kirchen, 1954; Kurz, 1927; Pfingst, 1921 & Stocker, 1934). Rarely myopia may be present at birth as a consequence of posterior scleral ectasia. Prematurity is also frequently associated with myopia. The most common form of myopia involves genetically predetermined abnormal ocular growth to late adolescence.[32] Before the onset of juvenile myopia, the premyopic eye in children with a family history of myopia already resembles the elongated eye present in myopia.[17] Before the start of formal grade-school education, a myopic shift is most likely to be attributed to a genetic predisposition from their myopic parents.[17] Likewise, family pedigrees have shown substantial refractive correlation between parent and offspring (Wold, 1949). Recently Zadnik et al. (1994) demonstrated an increased incidence of myopic ocular components in children with two myopic parents. This study suggested a strong hereditary component in the development of childhood myopia. Francis Young (1969) reported that while there was virtually no myopia in the parents and grandparents, there was a high incidence approximately 58% of myopia in the offspring. They dismissed

any hereditary factor in the development of myopia as there was no correlation between the refraction of the parents and those of offspring, while sibs of similar environment showed substantial correlation. From studies conducted in the United States and Europe, the best estimates of the prevalence of myopia among children of myopic parents are on the order of 30-40% when both parents are myopic, 15-25% when either parent is myopic, and 10% when neither parent is myopic (Goldschmidt, 1968; Ashton, 1985; Gwiazda et al., 1993). Thus there may be some predictive power in knowing the parental refractive error history. There is an increased incidence of myopia in children with two (compared to zero or one) myopic parents.[55] Children with higher degrees of myopia show a greater degree of hereditary influence in comparison to those with lower degrees of myopia. It has been found that the percentage of myopic parents increased with degree of myopia of children.[91]

4.1.3 EPIDEMIOLOGY OF MYOPIA

Since the mid-19th century a large number of demographic and correlation studies of myopia were carried out. Cohn (1867) carried out a classic investigation about the incidence of refractive errors among 10000 school children. Cohn and others observed that the number of myopes and the degree of myopia increased with age. Myopia mainly occurred after spending many years at school and the development of myopia was due to close work. This theory was widely accepted as school myopia. Michaels (1975) estimates that as many as 40% of the adult population in the United States have myopia in some degree.

Since then most investigations concentrated mainly on two theories a) hereditary and b) environmental. Those who favored the hereditary theory, believed that the differences among ethnic groups resulted from a genetic cause and that the increased incidence rate of myopia among school children was related not to school experiences but to age. In contrast, those who

believed the "use-abuse" theory stated that school experience was a significant factor in causing myopia.[58]

Many studies have been conducted regarding the prevalence of myopia and results differ depending on the definition of myopia, investigation methods, examination procedure, age of population, racial composition and socio-economic status. The socio-economic and clinical importance of myopia is as great as nearly 90% of glasses worn between the ages of 12-27 years are prescribed for this type of refraction (Bear & Richler, 1982). More than eighty percent of those reporting to school eye clinics have myopia. Harman (1936) gives a figure of 27% for Britain , Jackson (1932) 19.6% in the United States and Angel & Wissman (1980) 28.4% in males and 36.5% in females in the USA. Rasmussen (1936) reported a 70% incidence in young Chinese adults. Its prevalence in this group is about (15%) but in other countries pre-school children have far lower rates of myopia less than 2% (Zadnik et al, 1994).

In a detailed study of visual screening in the United States (1976) the National Eye Institute found myopia to be the fifth most frequent specific cause of impaired vision, the eighth most frequent cause of severe visual impairment, and the seventh most frequent cause of legal blindness.[2] Among causes of visual disability and blindness throughout the world, myopia is one of the most important causes. The expense related to optical correction and the complications that occur in its advanced forms make myopia a serious social and economic problem.[2] The estimated annual cost of myopia in USA is about 3.4 billion US$. Myopia in its usual form implies only a slight handicap and is correctable by glasses. On the other hand it can be of a progressive, degenerative type producing severe uncorrectable loss of vision.[11]

The prevalence of myopia in the normal term new-born is probably between 4% and 6%, although a literature review shows extremes of 1% and 25% (Curtin, 1985). Low myopia appears to resolve or decrease over the first few months of life. The incidence of myopia remains fairly stable over the first few years of life but shows an increase with advancing age through childhood.

This increase is seen at about the time children are beginning school and this has implicated near work and accommodation as a cause for the development of myopia. Studies of prevalence of myopia in adults show a wide variation (from 8% in Sweden to 52% in China) depending on the population and the age groups examined.[42] Myopia is common in Chinese population. According to Chen & Lin (1983) the prevalence rate of myopia was 46% for children aged 12 years and 78% for children aged 15 years. The rate of myopia among Hong Kong Chinese pre-school children increases with increasing age and exceeds 50% by age of 10 years.[49] Regarding the relative prevalence of refractive error it was seen that myopia was the common refractive error among the children. Among school children in Bangladesh, 47% are myopic, 19% are hypermetropic and 34% are astigmatic.[47] Similar findings are also available from India by Dutta et al. (1983) and in Japan by Majima et al. (1960). Myopia is quite patterned in its occurrence. Patterns reappear in many countries at widely different periods.[73] It is non-randomly distributed by age where it occurs (Slataper, 1950). It is rare among infants of industrial countries but in a birth cohort it increases in prevalence and severity through the end of adolescence and then stabilizes. Myopia has been found to be patterned in its occurrences in different races and ethnic groups. Blacks, whether in Africa or in US have been observed to have a low prevalence of myopia (Holm S, 1937; Callan P, 1875). Orientals (Rasmussen OD, 1936) and Jews (Kantor D, 1932) have been found often to have a high prevalence of myopia. Myopia is known to be closely associated in prevalence and severity with educational attainment. It has also been found sometimes to be positively associated with social class (Goldschmidt E, 1968), degree of urbanization of place of residence, and level of economic development of region or country of residence (Wangspa S et al., 1965 & Mann I, 1966). Some studies have also found females to be slightly more myopic than males (Goldschmidt E, 1968 & Baldwin W, 1967). Studied by Sheu et al. (1984) in kindergarten children found that 58.9% were hypermetropic, 39.8% emmetropic and 1.3% were myopic eyes.

The prevalence of myopia varies with age and place. In USA prevalence of myopia was 29.3% at the age of 12 and 33.2% at the age of 17.[21] In Taiwan another survey of school children showed that the prevalence of myopia increased from 4% at the age 6 years to 40% at the age of 12 years and at the age 15 the myopic was over 70%.[59] The prevalence was as high as 92.9% among University students.[37]

A high prevalence of myopia was found among Chinese, Japanese and Koreans, while a low prevalence of myopia was found among Indians[11], affecting 25% of most populations but up to 80% of school children. Heredity, environment or both may be the reason for high prevalence rate of myopia in Chinese.[92] The studies of Goldschmidt in 1964 discovered some associations between nearwork and prevalence of myopia. The prevalence was 11.8% in an office work group while only 4.3% was found in heavy work group.

In Taiwan 4% are myopic at the age of 6 years and 70% are at the age of 15 years. In Hong Kong 76% are myopic between the ages of 26-32 years.[2] The incidence of myopia was up to 51% for primary school children, 60% among high school and 85% among college students.[34] The prevalence rates were comparable with reports from Taiwan- 92.9% (Lin et al., 1989), Singapore- 83% (Chew et al., 1990), Poland- 44.5% (Muskaliski K, 1990) and India- 24.3% (Mukherji et al, 1979). Skeller (1969) had observed a high incidence of myopia in young Eskimo children: 9 out of 60 aged 2 to 4 years, and 29 out of 176 aged 5 to 9 years, had myopia of 0.25 D or more. Baldwin (1964) found that the incidence of myopia varied from a high of 17% for Chinese children to a low of 3% for Hawaiian children and for the Japanese it is 12%. It was found that in non-Jewish boys myopia increased from 14% at ages 8 to 9, to 28% at ages 13 to 14 whereas in Jewish boys it was 40% for all age group and was due to apparently genetic factors and not due to excessive amounts of close work.[63] Reber (1964) reported an incidence in Negro children of 8%, while in Caucasians it was 13%.

Rosner & Belkin (1987) found a correlation between myopia and years of schooling. The prevalence of myopia among those who had completed only eight years of schooling or less was 7.5% and the rate rose to 19.7% among those who had completed 12 years or more.[51]

There have been few studies done in Hong Kong on pre-school children. One such study was carried out by Chan and Edwards in 1991. The mean spherical equivalent for 570 subjects was reported as +0.63 D at an average age of 4.3 years. More than 50% of pre-school children became myopic by the age of 10 years.[49]

Lam and Goh studied 383 school children in Hong Kong and observed that the mean refraction was -0.09 D at the age 6 to 7 years, -1.31 D at the age of 12-13 and -2.05 D at the age of 16 to 17 years. The prevalence of myopia in these age groups was 31.4%, 58.1% and 58.8% respectively.[50]

4.1.4 RELATIONSHIP BETWEEN MYOPIA AND OPTICAL COMPONENTS

A classical work was published by Donders in 1864 on accommodation and refraction. He stated that the dioptric power of the eye was the result of the convexity of the cornea, position and focal distance of the lens and the length of the visual axis. Each of them may differ in the emmetropic eye and they may complement each other. The refractive state of the eye is usually determined by corneal curvature, anterior chamber depth, lens power and the axial length of the eye. Extensive studies have shown that corneal curvature, anterior chamber depth and lens power when sampled from the general population show a normal frequency of distribution. The frequency distribution of axial length, however does not conform to a normal distribution but rather shows a sharp peak at approximately 23 mm with a skew toward higher axial length.[57]

In emmetropic eyes, the statistical correlation between the axial length and corneal power was found to approach -1. A negative correlation of 0.5 between axial length and lens power and a positive correlation of 0.5 between

axial length and anterior chamber were also demonstrated. It was therefore questioned that there was a regulatory mechanism during ocular growth that caused the refractive components to combine in just such a way that eyes end up being emmetropic. In myopia, there is some disturbance of this process, brought about by hereditary factor, environmental factor or both.[57] Sorsby's work stands as the model for our current understanding of the evolution of ametropia, particularly myopia. He and his co-workers demonstrated conclusively in their study that an emmetropization effect was noted in distribution curves of refraction as a result of a correlation of corneal curvature to axial length. In ametropes of 4 D and above, this correlation appeared to break down, however. Their study also indicated that neither the lens nor the anterior chamber depth was an effective emmetropizing factor. In all of their investigations, the main finding was the high correlation of total refraction to axial length.

4.1.5 AXIAL LENGTH

Axial length is an important factor determining the refractive state.[25] The majority of low myopia cases are caused by increased axial length of the eyeball (Goldschmidt, 1981). If the axial length exceeds 26 mm both collagen and hyaluronic acid concentrations have been found to be approximately 50% lower than emmetropic eye.[33] Classical Sorsby studies led to the concept of axial length increase up to the age of 12-13 years and the same appears valid in Japan today.[35] An increase in the size of the eye during the early years of life is well documented. This growth have been divided into a rapid phase (birth to 3 years) with growth of about 5 mm called the rapid phase and a slow phase (3 to 13 years) of about 1 mm or 0.1 mm per year. It has been established that almost all childhood and adolescence myopia is due to axial elongation. Adult onset of myopia probably is caused by axial elongation (Curtin, 1985). An axial length increase is obvious together with a refractive change towards myopia,

while corneal curvature remained rather stationary.[36] Corneal curvature was found to play only a minor role in the determination of refraction and the measurements of axial length paralleled the degree of myopia.[37] In the young Chinese population there is a 2 mm shift in the axial length (normal = 23 mm) of the eyeball to a mean value of 25 mm (Sheu MM, 1982). The primary role of axial length in human refractive error was clearly established by Stenstorm (1948). The axial length is the dominant factor, with changes in it being correlated with growth changes in the other elements in such a way that the resultant effect is a relatively stable refractive state.[78] A high correlation was found between the refraction and axial length. The more myopic eye of an anisometropic eye-pair invariably had the greater axial length.[82] Axial length increases 0.1 mm each year and lens power decreases by 0.2 D (Lin et al, 1990).

4.1.6 VITREOUS LENGTH

Vitreous elongation continued until the age of 10-12 years and a similar curve was obtained regarding axial length. That is, normal growth of the axial length stops at about 10 to 12 years. After 10 years of age the axial length changes only little in normal eyes.[38] The average variation of axial distances per 1 D of myopia development is as follows: an axial length increase of 0.24 mm, a vitreous length increase of 0.29 mm, an anterior chamber depth increase of 0.076 mm and a lens thickness decrease of 0.073 mm (Kamiya, 1986). Consequently the refraction will be dependent on the length of the ocular axis, that is mainly on the length of the vitreous cavity.[38] There is a high correlation between the refraction of the eye and length of the vitreous cavity. Myopia is usually created by a lengthening of the vitreous and a very strong correlation was found between refraction and vitreous length since it is not affected during corneal indentation or applanation .[81,97] Even very low myopia shows elongation of the vitreous length from the age of 10 or so. Vitreous

length is the most important single parameter in axial myopia.[39] If near work induces myopia without ciliary spasm or lasting lenticular myopia, another mechanism would be the elongation of the vitreous chamber.[40]

From the Japanese population the following are summarized from studies (A Hosaka, 1988)

i) The refractive condition in each person is mainly determined by the axial length.

ii) The axial length of the eye depends mainly on the length of vitreous cavity.

iii)The anterior part of the eye that is from the corneal surface to the posterior surface of the lens may differ from individual to individual.

iv)However, the differences found are rather small and anterior segment axial distances do not play an important role regarding refractive value.[38]

4.1.7 CHANGES OF REFRACTION IN PRE-SCHOOL CHILDREN

Refraction goes through various changes during the life span of an individual and these changes depend on age. It was earlier believed that all infants were hyperopic at birth but was later determined that as many as 20% to 30% of new-born were myopic (Goldschmidt, 1969; Mohindra, 1980; Cook & Glasscock, 1951) and that the incidence is higher in premature infants (Scharf J et al., 1975). Considerable changes in refraction occur during the first year of life. In the first seven years of life a child becomes, on the average, slightly more hyperopic or less myopic (Brown EVL, 1938 & Slataper FJ, 1950). After seven years of age, the refraction change begins to reverse toward less hyperopic or more myopic. This change is most rapid between the ages of 10 and 14 years (Brown EVL, 1938 & Slataper FJ, 1950). Small changes are still recorded between the ages of 14 and 20 years, after which time changes are few (Brown EVL, 1938 ; Slataper FJ, 1950 & Sorsby A et al., 1970). Studies found that children who are hyperopic tend to retain the same refraction for years or to experience minimal changes, becoming less hyperopic. As for

children who are or have become myopic, in one year the changes can be even 2 diopters toward more myopia.[41] According to this study, the change of refraction in hyperopic children proceeds at a much slower rate than in myopic children.[41] It is known that a large amount of axial growth and corneal flattening takes place over the first few years of life.[42] Although it has been shown that there is a higher risk of myopic progression in children with myopic fundus changes, with intraocular pressure above 16 mm Hg, and with myopia greater than or equal to 3 D (Jensen H, 1991), for the most part it has been assumed from the increasing prevalence of myopia seen with age in children, that myopia tends to progress. This may not, however, be the case for any individual child.[42] Myopia increased from 6% in the age group 5 to 10 years to a peak level of about 35% in adults aged 20-40, with a subsequent decrease towards old age, where hypermetropia showed a significantly increasing share.[43] Hypermetropia of the young child decreases, myopia increases during adolescence and early adult life, and towards senescence a hyperopic drift is seen.[43] The myopic infant is less likely to develop amblyopia and strabismus and low degrees of myopia may be left uncorrected in infancy. Above -3 diopter, the myopia should be fully corrected. In the school age child with low degrees of myopia are corrected when reduced distant vision interfere with school work or games.[44] Verlee (1968) suggested that an increase in myopia of school children may be expected with increasing near tasks.

In Hong Kong most children aged from 3 to 6 years attend kindergarten and start writing simple English and Chinese words. They commence primary school at the age of six or seven, with long hours in school and doing homework. Hence, an early age to commence study, long hours of schooling and nearwork particularly with Chinese characters might contribute to the high prevalence and severity of myopia in this population. Non-cycloplegic refraction data in the first year of life reveals a shift from myopic readings in the early months to emmetropic readings by 6 months (Mohindra & Held, 1981). In contrast, cycloplegic refraction were found to shift from myopia or emmetropia

in the first months to more hyperopic readings by 6 months of age (Abrahamsson & Sjostrand, 1992; Schaloj-Delfos et al., 1992; Wood & Hodi, 1992). Children who had negative spherical equivalents as infants on average never reached the same level of hyperopia as those who had positive spherical equivalents.[56] Studies show that the children who develop school-age myopia can be predicted from their infantile manifest refraction. Refraction in older children can be predicted from their earliest manifest refraction, although the refraction at 1 year is a better predictor than at 3 months. The high correlation throughout childhood demonstrate that individuals tend to remain at their initial positions in the distribution of manifest refraction, even though its dispersion reduces during the pre-school years.[56] Childhood myopia progression is usually due to axial elongation, which is not compensated by reductions in corneal and crystalline lens power (Tokoro & Suzuki, 1969; Sorsby A, 1979; Fledelius HC, 1982). It clearly shows that the changes of refraction in hyperopic schoolchildren occur much more slowly than in myopic children of the same age.[61]

4.1.8 DEVELOPMENT OF MYOPIA

Considerable changes in refraction occur during the first year of life. In the following one or two years, the most striking change is the decrease in astigmatism (Ingram & Barr, 1979; Atkinson et al., 1980; Gwiazda et al., 1984 ; Howland and Sayles, 1984, Edwards M, 1991). In the first seven years of life, a child becomes, on the average, slightly more hyperopic or less myopic (Brown EVL, 1938; Slataper FJ, 1950). After seven years of age, the refractive change begin to reverse toward less hyperopia or more myopia. This change is more rapid between the ages of 10 and 14 years (Brown EVL, 1938; Slataper FJ, 1950). Small changes are still recorded between the ages of 14 and 20 years, after which time changes are few (Brown, 1938; Slataper FJ, 1950; Sorsby & Leary, 1970). It has been suggested that myopia that develops in earlier school

years (i.e., before puberty) could progress faster than that which develops later (Rosenberg & Goldschmidt, 1981; Fledelius, 1981). If myopia is already present in infancy and in early childhood, then the influences involved at this stage are likely to be hereditary rather than environmental.[68] The human eye undergoes extensive growth in the postnatal period. The increase of approximately 7 mm in axial length from birth to adulthood requires a reduction of approximately 30 diopters of total refracting power to maintain an emmetropic state (Bennett & Francis, 1962). After 5 to 6 years of age, the axial length increases approximately 1 mm to its adult length.[62] Young (1981) noted that myopia appeared to develop in two stages. The first stage was the development of a form of prolonged accommodation giving a temporary change in lens thickness, followed within less than 1 year by an increase in the size of vitreous length. Raviola & Wiesel (1985) observed in animal experiments that axial myopia developed in the lid-sutured eye, and the corneal curvature, anterior chamber depth, lens power of the lid sutured and the control eye were same. The same result was found in opaque cornea made by polystyrene beads, proved that this was not due a to temperature effect in the experimental eye. The myopigenic effect was greatest if the eyes are sutured at birth, but had no effect in the adult eye. Destruction of the visual cortex had no effect on the development of lid-sutured myopia, indicating subcortical or ocular control of eye growth.[57] In the rhesus monkey, neither the continuous instillation of atropine nor the transection of the optic nerve prevented the development of lid-sutured myopia, indicating that myopia develops via intraocular mechanisms independent of central nervous system feedback or ocular accommodation. Half lid-sutured in the stump-tailed monkey showed that accommodation and central nervous feedback played a partial role in the development of myopia. A minus lens makes a chicken develop axial myopia, whereas a plus lens stops the normal elongation of the eye, making the eye hyperopic. These effects appear to be independent of ocular accommodation (Schaeffel et al., 1990). In experiments where the temporal portion of the

chicken's visual field was restricted by the use of an occluder, the animal developed an eccentric expansion of the globe, causing an axial myopia in the occluded visual field. In the unoccluded portion of the globe was emmetropic.[57] The focal length along eccentric visual axes of the chick eye change to correspond with the visual environment in the portion of its visual field (Miles & Wallman, 1990). Raising chickens in a low-ceiling environment caused the development of myopia because of eccentric expansion of the vitreous chamber in that portion of the eye corresponding to the superior visual field. Posterior expansion in the experimentally induced myopic eyes is the result of scleral growth (as opposed to scleral stretching) and this growth occurs primarily in the inner as opposed to outer sclera (Christensen et al., 1990; Rada et al., 1990 & Wu YR, 1990). Furthermore, this growth appears to vary in different regions of the sclera, being greatest at the posterior pole (Christensen et al., 1990). McBrien et al. (1989) showed that the lathyritic agent aminoproprionitrile, which weakens newly formed collagen, can increase the axial elongation and cause further thinning of the posterior sclera in experimental myopia. Although periods of accelerated growth may be much shorter, the growth processes that take place over weeks in the chick eye take place over one or two decades in the human eye. This extra period of time in the human adds to the difficulty of trying to solve the role of environmental and genetic factors in the development of myopia. In pathologic myopia there may be a abnormal collagen metabolism that leads to a progressive asymmetric stretching and thinning of sclera. Intraocular pressure, although important in normal ocular development and possibly in the development of all types of myopia, may play a more important role in this particular form of myopia. Abnormal susceptibility to normal or increased intraocular pressure may contribute to the posterior scleral expansion and staphyloma development. In the early stage of myopia development the anterior-posterior diameter of the eye elongates.[64] An early onset means a progression which continues over a longer time, while a later onset implies a slower progression for a shorter time.

The mean progression of myopia seems to continue at least up to 20 years of age for those whose myopia starts before the age of 18.[72] Hirsch (1961) found that during the first six years of school between 84 to 90 per cent of the children show a refraction change in a linear fashion. During this same period, between 6 and 8 percent of the children show a refraction variation in a curvilinear fashion, the curve being convex upward. Van Alphen (1961) proposed that the growth of the lens and cornea are influenced genetically but that accommodative activity influences growth of the axial diameter and thereby allows maintenance of emmetropia during development. When accommodation is increased ciliary muscle activity facilitates axial growth of the globe, whereas when accommodation is relaxed axial growth is impaired.[77]

4.1.9 EMMETROPIZATION

The development of refraction is the result of a complex interaction between the growth of various ocular tissues. Many hypothesis regarding emmetropization have been postulated but the mechanism still unclear. This may represent a series of polygenic interactions where the phenotypic expression relies on environmental conditions. In a study by Troilo and Wallman (1991) both the myopic and hypermetropic eye were initially larger than normal yet growth of the vitreous chamber stopped in eyes compensating for myopia and continues in eyes recovering from hyperopia, regardless of the size of the eye. Refractive state rather than eye size guides the eye toward emmetropia.[90] Emmetropization depends upon the coordinated growth of the cornea, anterior segment, lens, and vitreous chamber. Growth of the anterior segment reduces the total optical power of the eye by decreasing the curvatures of the cornea and lens and increasing the depth of the anterior chamber. Corneal power, lens power, anterior chamber depth and axial length (along with the respective indices of refraction of the different media) combine to determine the refractive state of the eye. Statistical analysis has shown that

significant correlation exist between the refractive components in emmetropic eyes of adults. Through infancy and childhood, these components undergo changes which result in the average refractive state changing from mild hyperopia to emmetropia.[42] Since a preponderance of emmetropia is seen in the adult population, there appears to be a developmental process (so-called 'emmetropization') which results in growth of the refractive tissues toward emmetropia. Largely on the basis of experimental studies reviewed elsewhere (Whitmore W G, 1991; Schaeffel F & Howland H C, 1988), it is felt that emmetropization is an active process and not just a passive independent growth of the ocular tissues. An emmetropization process occurs during the first 4 to 5 years (Mohindra & Held, 1981). During this period the manifest refraction of most children tend to converge on a slight degree of hyperopia. Although the mechanism of emmetropization is unclear, there are some suggestive leads. To some degree the overall uniform growth of the eye reduces ametropia (Wallman & Adams, 1987). Infants who suffer a lack of visual feedback owing to pattern deprivation tend to develop axial myopia, which suggests an altered process of emmetropization (Rabin et al., 1981). Most of the infantile astigmatism, which is corneal (Howland & Sayles, 1985), is greatly reduced or eliminated by 5 years of age, similar to the time period for the overall emmetropization process. Usually emmetropization arises from accommodation, but when too much accommodation occurred it would be distorted to the myopic side resulting in significant myopia.[88]

4.1.10 SEX

Both sexes are equally affected with lower degrees of myopia but females are more prone to the higher degrees and to degenerative changes.[7,5,23] There was between 52% and 67% penetrance in the dominant variety, females appearing to have a greater degree of expressivity.[3,8] For the most part pathologic myopia is an autosomal recessive disease. The female

has an increased genetic penetrance of high myopia.[8] Corneal curvature, anterior chamber depth, vitreous length and axial length are larger for male than female.[25] The prevalence of myopia is higher in females (17.6%) than males (15.3%). While the prevalence of mild myopia was higher among the males, and moderate to severe myopia was higher among females.[26] The ratio of females to males is 1.2-1.5 : 1. This may be related to the girls who have higher degree of sexual maturation at this age.[10] In general myopia starts earlier in females than males. Myopia has been found to be more common among girls in the Manchurian Railway Study (McLaren, 1961) which showed a prevalence of 25.4% for girls and 18.7% for boys, and also noted the results of a study among Japanese children in which girls showed a greater frequency of myopia 15.5% to 10.1%. In Goldschmidt's (1968) study the prevalence of myopia among girls was significantly greater than the boys ($p < 0.001$). Sharp increases of myopia usually occurs between the ages of 12 to 14 years and may be due to have a greater effect with early menarche (Gardiner, 1954) and usually amount of time spent on reading and close work greater among the girls.[96] According to Laatikainen et al. the annual incidence of retinal detachment is slightly greater for males than for females in all age groups but the difference was not statistically significant. Of the phakic eyes 44.6-57.4% were myopic (-1.0 D or more). Myopic detachments composed 34.5-45.7% of all detachments and the percentage varied from 70% in the age group of 20-39 years to 20% in the age group of 60 years or older.[27] In earlier studies males have been found to be affected more frequently than females (Duke-Elder, 1967). In the present study the proportion of males to females corresponded with that in the general population except in the youngest age group where males had a preponderance of 3:1 mainly because of the high incidence of traumatic detachment in boys.[27] In non-traumatic retinal detachment there was a small sex difference with a higher incidence among females. At lower ages the morbidity from retinal detachment was higher in males than in females.[28] The distribution of refractive errors in the non-traumatic retinal detachment

cases did not show any sex difference. In patients less than 60 years of age the incidence rates were higher in females than in males.[28] When combined for all ages prevalence rates were significantly less for men than for woman.[21] From the age of 25 to 80 myopia seems to be equal in women and men.[22]

4.1.11 RISK FACTORS

Many factors such as sex (Goldschmidt 1968; Aine, 1979; Richler & Bear, 1980; Angel & Wissman, 1980), close work/educational level (Tscherning 1883; Yog 1975; Richler & Bear, 1980), hormones (Balacco-Gabrielli & Tundo, 1981) and dietary aspects (Gardiner, 1958; Lane, 1981) may play a role in causing myopia. The pathogenesis of myopia in humans is a complex and multifactorial process. Hereditary and environmental factors are both important in determining the ultimate refraction of the eye, although their relative importance has yet to be elucidated. Numerous studies suggest that heredity, environment or both are involved. Myopia may be associated with hereditary retinal or vitreoretinal disorders, e.g., retinitis pigmentosa, choroidermia, gyrate atrophy, Stickler's and Wagner's syndrome (Curtin, 1985), cone dystrophy (Francois, 1974). Heredity studies in myopia have shown autosomal dominant and autosomal recessive types of inheritance.[84] There are two theories which have related myopia and education level. The biological theory described by Sofaer and Emery (1981) in a study of British members of Mensa showed an association between high IQ and myopia. Evaluation of scholastic achievement test scores by Heron and Zytkoskee confirmed that persons with poor visual acuity perform better on tests than individuals with normal acuity. Peckham et al. found that superior educational attainments were already apparent before the onset of myopia as found in 7 year olds. The biological theory views myopia as the result of genetically determined characteristics of eye tissues. It predicts that either age from birth or age from puberty explains any tendency for myopia to appear and progress among children or adolescents. It implies that the

myopia genes are inherited with those for intelligence, hence the correlation of myopia with education.

Bear, Richler and Burke found that refraction was consistently correlated with nearwork from ages 5 to 60 even after adjustments for age and sex. Large amounts of nearwork in childhood contribute to the prevalence of clinical myopia.[22] Angel and Wissman noted that when one controlled for education, age was not related to increased myopia. Young et al. (1969) and Woonruff & Somek (1977) found that the introduction of formal education in Eskimo and Amerind populations has dramatically increased myopia. In utero ocular growth proceeds according to poorly understood genetic factors. Environmental disturbances at this time (i.e. maternal illness) may upset the normal relationships between the refractive components resulting in the presence of myopia at birth or in conditions which lead to the development of myopia in early childhood. Congenital or developmental myopia can be seen in association with systemic disease, with ocular disease alone, or as an isolated finding. The mechanism by which prolonged accommodation, convergence and/or near visual output over long periods of time might cause an increased prevalence of myopia seen is still debated. Coleman (1970) has postulated an aqueous-vitreous pressure gradient (pressure greater in the vitreous) during accommodation which presumably could result in increased stress on the sclera with consequent stretching and elongation of the globe. The factors which influence ocular development during the period of growth are as yet uncharacterized, although there have been numerous studies supporting hereditary determinants of myopia (Wold, 1949; Sorsby., Sheridan & Leary, 1962; Curtin, 1985; Lin & Chen, 1987; Teikari., O'Donnell., Kapiro & Koskenvuo, 1991; Yap., Wu., Liu., Lee & Wang, 1993; Zadnik., Satariano., Mutti., Sholtz & Adams, 1994). Twin studies, in particular, have provided strong evidence for the inheritance of myopia.[53] That diet may have some effect on myopia is confirmed by the work of Walkingshaw (1964) and Gardner (1964) in England. Three risk factors for myopia were evaluated to predict myopia: a)

refraction in infancy b) refraction at school entry and c) parental history of myopia.[55] The basic tenets of accommodation theory are that the accommodation and/or convergence create force on the sclera and a resultant increase in intraocular pressure. This higher pressure would then be poorly resisted by the sclera, resulting in expansion, excessive ocular length and myopia (Van Alphen, 1961; Young, 1975). Destruction of the visual cortex had no effect on the development of lid-suture myopia, indicating subcortical or ocular control of eye growth. Conflicting results for tests involving accommodative blockade and optic nerve transaction were found in the two different species monkey used in the experiments. In the stump-tailed monkey the half lid-sutured myopia indicating that accommodation and central nervous system feedback played a partial role in the development of myopia in this species.[57]

Heredity is an important factor in the development of myopia and demonstrated clearly by the high concordance of refraction between siblings in twin studies. The correlation, which approaches unity for monozygotic twins, is still significant between siblings of dizygotic twins, but falls to zero in matched pairs. Genealogic studies have shown both autosomal dominant and recessive modes of inheritance for myopia in certain pedigrees, but the majority of studies fail to show any strict pattern of heredity. A polygenic transmission, therefore, is presumably the predominant mode of inheritance. The polygenic nature of myopia inheritance and the complexity of myopia development will continue to impede attempts to relate genotype to myopia phenotype. According to Hirsch (1969) & Ashton (1985) children who are myopic are more likely to have parents who are also myopic. The greater the child's myopia, the more likely the parents are also myopic. When both parents are myopic, the prevalence of myopia in the offspring is at least three times higher than when neither parent is myopic. It is well known that children of Chinese or Japanese descent have a high prevalence of myopia. At the high school level it is not unusual to find myopia in over 60% of these children (Lin et al., 1988; Hosaka, 1988; Edwards

& Lewis, 1991). However, a recent study suggests that myopia found in infancy is probably inherited in an autosomal recessive manner (Edwards & Lewis, 1991). The theory described by Steiger (1913), the characteristics of each components of the eye which affects spherical refraction are genetically determined and in the population independently and normally distributed (Gaussian distribution) with means that produce emmetropia.[75]

It is generally accepted that environment plays a role in the development of myopia on the basis experimental evidence in animals and because of strong clinical correlation with situations similar to these experimental conditions in humans. Isolating different environmental influences in myopia development and the different effects they have on the various components of refraction has been a particularly perplexing problem. The strong association of myopia with a history of near work over prolonged periods implicates ocular accommodation as a factor in the development of myopia. That atropine does not stop myopic progression in all patients indicates other factors are involved.[57] A study of mildly myopic schoolchildren showed that myopic progression could not be stopped by the use of bifocals or by reading without spectacles (Pärsssinen O et al., 1989). On the contrary, this study found a trend toward less myopic progression in those children who continuously used their full spectacle correction. The degree of myopia found in population surveys is influenced by age, sex, ethnicity, level of education, social class and level of urbanization (Angle & Wissmann, 1980; Curtin, 1985). These effects can be explained by postulating that nearwork, particularly reading, is one of the prime factors causing myopia.[76]

In its most common form, the use-abuse theory of myopia states that continual near-work, such as reading, causes myopia. Virtually all use-abuse theorists share the view that near-work results in the tensing of the extraocular muscles and that this tension over time makes the eye permanently more myopic. The use-abuse theory explains the epidemiology of myopia in terms of the amount of near-work people in different social categories do.[73] In this

theory, continual intensive accommodation is said to elongate the choroid and sclera along the anterior-posterior axis of the eye and thus to cause myopia (Dunphy E, 1970; Young F, 1970). Studies since the 19th century have continued to show increases in the incidence and severity of myopia during the school years.[75]

A role for increased intraocular pressure in the development of myopia is suggested by cases of unilateral congenital and juvenile glaucoma where the affected eye become more myopic. Coulombre (1956) studying embryonic chick eyes, demonstrated stunted scleral development (despite normal growth of choroid and retina) in eyes with intraocular pressure lowered to zero, supporting a role for intraocular pressure in normal ocular development. Pruett (1988) reviewed the clinical and experimental evidence in favor of a primary role for increased intraocular pressure (or abnormal susceptibility to normal intraocular pressure) in the development of myopia. Transient and repeated elevation of intraocular pressure may be caused by different actions of the extraocular muscles (e.g., convergence, downgaze), accommodation, and eyelid squeezing. Other factors may include dependent posturing of the head may result in hypostatic congestion in the eyes i.e. increased venous pressure in the choroid and ciliary body (Levinsohn, 1931 & Mohan et al. 1977). These intraocular pressure elevations may cause irreversible stretching of the sclera in genetically or pathologically susceptible eyes over time.

CHAPTER 5

5.1 METHODS

5.1.1 SUBJECTS

A kindergarten located at the Sha Tin, New Territories was randomly selected. Details of the proposed study were explained to parents and school teachers. All subjects who could complete the questionnaire and undergo the complete ocular examination, which included cyclopegic refraction and biometric measurements were included in the study. Exclusion criteria were: incomplete questionnaire or ocular examinations, cases of squint or amblyopia and over age.

The reason for refusal to participate were: a) Parents were too busy to fill out the questionnaire, b) Parents worried about side effects of cycloplegic eye drops and c) Parents thought that their children were normal and there no particular reasons to participate in the study. It was found in these cases that all of them had normal vision, with visual acuity of 20/30 or better in the selected subjects.

The number of overage was 32, strabismus 3, amblyopia 2, failed refraction and A-scan after dilatation was 4. The medical and surgical history was only included allergic rhinitis, mild respiratory distress and tonsillitis.

A total of 237 childrens (girls =114 and boys =123) and their parents were included in the study. No pattern emerged to suggest that self-selection was causing a bias in the distribution of refractive errors in the sample.

All studies were on school grounds during regular school hours. The eye examination included visual acuity assessment, refraction & keratometry before and after dilatation, A-scan for anterior chamber depth, crystalline lens thickness, vitreous chamber depth and axial length.

5.1.2 PROCEDURE

5.1.2.1 QUESTIONNAIRE

The questionnaire was composed of four parts: a) general personal information, b) major illness and ocular history, c) mother's and father's personal information, school achievement, history of myopia, age starting wearing myopic glasses, family history of high myopia and d) amount of near work, both time and distance in school and home, time of exposure to TV, computer, video-game and game-boy (Appendix I-II). Diopter-hours was measured as hours per week, as reported by the parents and school teachers in activities such as writing, reading, and TV watching. A single week's diary of the child's activities were recorded.

5.1.2.2 EYE EXAMINATION

A standardised eye examinations were performed which included: i) visual acuity, ii) autorefraction iii) keratometry and iv) A-scan biometry (Appendix III-IV).

I) Visual acuity: The visual acuity with or without glasses was measured by E optotype (Sheriden-Gardiner VA Chart) in both eyes separately at a standard (6 m) distance and with pinhole.

II) Auto-keratorefraction: Refraction with and without cycloplegia were measured by Topcon KR 7100 auto-keratorefractometer. Auto plus fogging procedure was used during autorefraction. Each eye was measured three times including after dilatation. Tropicamide 1% and cyclopentolate 1% were used three times at 10 minutes interval for dilatation and the eyes were examined after 30 to 45 minutes. The refractive values were taken as the spherical equivalent (SPEQ) in diopters (D). SPEQ = spherical value + ½ of cylinder

value. Myopia was defined as a spherical equivalent greater than -0.50 D, emmetropia from -0.50 to +0.50 D and hyperopia was greater than +0.50 D.

III) Keratometry: Keratometry of both eyes was performed by the auto-keratorefractometer (Topcon KR 7100) with or without cycloplegia.

IV) A-scan biometry: Anterior chamber depth, crystalline lens thickness, vitreous chamber depth and axial dimensions were measured by A-scan ultrasound with Stortz Compuscan LT V2.00 @ 19993 with a hard probe (to reduce likelihood of probe compression) which has a central fixation light. The ultrasound velocity of was 1550 m/sec. Speed for lens was 1640 m/sec. Prior to this measurement one drop of proparacaine hydrochloride 0.5% was instilled after full dilatation of pupil. After proper asepsis three readings were recorded in each eye, and average values were calculated. Antibiotic drops were instilled after examination.

5.2 DATA ANALYSIS

All the data were computerised. Means and standard deviations (SD) of quantitative data were calculated. Diopter-hours were calculated as sum of visual tasks in school, home and exposure to TV (time x 1/distance) hours per week. All the visual tasks were analyzed first individually of writing, reading and TV (duration and distance), then combined time of writing and reading, distance of writing and reading both in school and home. TV watching (duration and distance) were calculated separately. The results were analysed with independent sample t-test, multivariate analysis of variance, Levene's test for equal variance, correlation coefficient, Pearson Chi-square test of independence, multiple regression analysis, Mann-Whitney U, K W One-Way Anova, Scheffe test ,Wilcoxon Rank Sum W test, reciprocal (Duration x 1/Distance) and analysis of covariance using the statistical package SPSS/PC+. Parental myopia and grade of education were categorical variables, others were treated as continuous variables.

CHAPTER 6

6.1 RESULTS

The participation rate were 83.73%. After applying exclusion criteria there were 114 girls (48.10%) and 123 boys (51.90%) whose age ranged were 36 to 83 months (mean 59.43 months; see Figure 1). Twelve children (5.06%) with myopia > 0.05 D were excluded from analysis.

6.1.1 THE DISTRIBUTION OF REFRACTION

In this study, myopia was defined as SPEQ more than -0.5 D, emmetropia -0.5 D to +0.5 D and hyperopia more than +0.5 D. The distribution of refraction including spherical equivalent (SPEQ) is shown in Table 1(a).

The refraction in RE was 0.86±0.84 D (Mean±SD) and values ranged from -1.50 to +5.00 D. The percentage of myopia among the subjects was 4.22%, emmetropia 28.69% and hypermetropia 67.09% (Table 8).

The refraction in LE was 0.82±0.95 D (Mean±SD) and values ranged from -2.00 to +5.00 D. The percentage of myopia among the subjects was 6.76%, emmetropia 31.22% and hypermetropia 62.02% (Table 1b). The RE & LE are shown in Figure 2.

The analysis shows there was no significance difference between the visual acuity in the right and left eye (t-test: $P > 0.05$). Table 2 shows the performance of visual acuity.

The right eye would be considered for analysing the correlation between the different variables for the following reasons a) there was a high correlation between the refractive value in the right and left eyes ($r = 0.3641$ and $P < 0.001$); b) the statistical assumptions of independence would be violated if both eyes are used indistinguishably in the same analysis and c) there are some publications which suggested to study RE only (Midelfart A et al., 1992;

Edwards M, 1991; Lam & Goh, 1991; Bear et al., 1981) and all the figures given are for the right eye unless otherwise stated.

6.1.2 THE RELATIONSHIP BETWEEN AGE WITH REFRACTION AND OPTICAL COMPONENTS

The relation between age and refraction

The analysis shows there was no significant relation between age and refraction where r = -0.0390 and p = 0.550.

The refractive components including corneal curvature (CC), axial length (AL), anterior chamber depth (ACD), crystalline lens thickness (LT) and vitreous body length (VL) have been analysed with different variables. The mean of axial length in RE was 21.97±0.79 mm (range 19.67-23.99 mm) and in the LE the mean was 21.91±0.88 mm (range 18.04-23.98 mm). The RE & LE distribution shown in Figure 3. The correlation between the right and left axial length was significant (r = 0.1560 & P < 0.05). In the analysis for the relationship either between ocular refraction and optical components or with different variables, the right eye of the subjects have been used for all after pupil dilatation and all the figures given are for the right eye unless otherwise stated.

Relation between age and axial length (AL)

For the right eye the axial length was 21.97±0.79 mm (range value were 19.67 to 23.99 mm). The axial length had a strong correlation with age (r = 0.2347 and P < 0.001) and the distribution is shown in Figure 5 and described in Table 4 and 10.

Relation between age and vitreous length (VL)

The vitreous length in the subjects was 14.99±0.67 mm (range value were 13.44 to 17.99 mm shown in Figure 4). There was a high correlation between age and vitreous body length where (r = 0.2245 and P < 0.001). The distribution is shown Figure 6. Relation shown in Table 6 and 11.

Relation between age and lens thickness (LT)

The lens thickness in the subjects was 3.79±0.35 mm (range value were 3.03 to 5.5 mm). There was no correlation between age and lens thickness (r = 0.0490 and P = 0.453).

Relation between age and anterior chamber depth (ACD)

The anterior chamber depth in the subjects was 3.18±0.36 mm (range value were 1.7 to 4.04 mm). There was no correlation between age and anterior chamber depth (r = 0.0516 and P = 0.429).

Relation between age and corneal curvature (CC)

The corneal curvature in the subjects was 44.16±1.5 D (range value were 40.50 to 49.37 D). There was no correlation between age and corneal curvature depth (r = 0.0008 and P = 0.991).

6.1.3 RELATION BETWEEN SEX AND REFRACTION

Relation between sex and refraction

The number of female subjects was 114, refraction among the girls was 0.95 ± 0.77 D in the RE. The number of male subjects was 123 and the refraction was 0.77±0.90D in the RE. Multivariate analysis of variance shows the two groups were not significantly different where t = -1.58 & P = 0.116. Table 7 and 8 show the distribution of refraction among males and females.

6.1.4 RELATION BETWEEN REFRACTION AND OPTICAL COMPONENTS

Relation between refraction and axial length

The axial length was 21.97±0.79 mm. Analysis shows that refraction and axial length were highly correlated where r = -0.4396 and P < 0.001. Figure 7 shows the distribution of refraction and axial length.

Relation between refraction and vitreous length

The vitreous length was 14.99±0.67 mm. The refraction and vitreous length were highly correlated where r = -0.3942 and P < 0.001. This has been shown in Figure 8.

Relation between refraction and lens thickness

The lens thickness was 3.79±0.35 mm. There was no relation between refraction and lens thickness where r = -0.0401 and P = 0.539.

Relation between refraction and anterior chamber depth

The anterior chamber depth was 3.18±0.36 mm. There was a negative relation between refraction and anterior chamber depth where r = -0.1990 and P = 0.002.

Relation between refraction and corneal curvature

The corneal curvature was 44.16±1.5 D. There was no correlation between refraction and corneal curvature where r = -0.0304 and P = 0.641.

6.1.5 RELATION BETWEEN REFRACTION AND VISUAL TASKS

The relationship between refraction and environmental factors i.e. visual task has been studied among the subjects. In the investigation the school teachers and parents were asked to record the hours spent in visual activities which included writing, reading, drawing/game, TV, computer, video-game and gameboy (Appendix I-II). They were also asked about the working distance and size of the instruments. According to data collected from the questionnaire and examination sheet, the relation between refraction, axial length, vitreous body length and diopter hours i.e., visual task (both duration and distance in school and home) have been analysed in the following ways (Distribution between visual task, SPEQ, AL and VL have been shown in Table 12-23):

Relation between refraction and working time

The relation between refraction and working time such as total writing time in school and home was 389.89±189.69 min/wk (range 60-1385 min), the total reading time was 203.60±164.60 min/wk (range 20-1165 min), the total TV watching time was 831.06±536.18 min/wk (range 10-3060 min). The analysis shows that there were no correlation between refraction and working time, $p > 0.05$.

Relation between refraction and sum of writing & reading (time)

The analysis shows the total time of writing & reading was 593.49±306.34 min/wk (range 80-2550 min). There were no significant correlation found where r = -0.0071 & P > 0.05.

Relation between refraction and working distance

The writing distance in school was 22.89±7.61 cm (range 10-40 cm), at home was 25.00±7.89 cm (range 10-50 cm), the reading distance in school was 25.13±5.43 cm (range 15-58 cm), at home was 27.11±7.46 cm (range 10-53 cm), the TV watching distance was 276.91±58.29 cm (range 175-380 cm) in school, at home was 198.61±71.34 cm (range 30-459 cm). There was no significant correlation found between working distance and refraction, p > 0.05.

Diopter-hours were calculated as the sum of near work in school, home and exposure to TV (time x 1/distance) hours per week. The summation represents the measurement of child's visual activities according to the amount of accommodation in diopters (D) required to perform them. According to the formula {3 x (writing time + reading time) + 2 x (TV game time) + TV time} (Zadnik et al., 1994) controlling for age, no relation was found between diopters-hours and children's refraction where p > 0.05. The present study uses the actual reported data, in contrast to Zadnik's formula where the fixed distance have been used.

Relation between diopter-hours and refraction

The analysis shows the diopter-hours was 30.37±15.52 hours (range 7-100 hours) in a week. There was no significant correlation found where r = -0.0531, P = 0.43. Diopter-hours of school and home have been described in Table 24-26.

Table 47 & 48 represent ocular components and refractive error data on the entire sample. The results show the axial and vitreous length increase with age. The estimate of the proportion of the variance explained by the models (SPEQ, CC, ACD, LT, VL, AL & LP) and controlling for 3 variables (age,

diopter-hours and parental history) as a function of parental myopia and of visual task as measured by diopter-hours. Since in these method no R^2 were available, Wilks Lambda test has been used to summarize all the models. No relation was found between diopter-hours and refraction (r = -0.0166, p = 0.799).

6.1.6 RELATION BETWEEN OCULAR COMPONENTS AND VISUAL TASKS

Relation between axial length and working time

The total writing time (school and home) was 389.89±189.69 min/wk (range 80-2550 min), the total reading time (school and home) was 203.60±164.60 min/wk (range 20-1165 min), the total TV time (school and home) were 831.06±536.18 min/wk (range 10-3060 min). No significant correlation (P > 0.05) was found between axial length and working time.

Relation between axial length and sum of writing & reading time

The writing and reading time (school and home) was 593.49±306.34 min/wk. There was no significant correlation between AL & reading and writing time where r = 0.1230 and p = 0.06.

Relation between axial length and working distance

The writing distance in school were 22.89±7.61 cm, at home was 25.00±7.89 cm, the reading distance in school was 25.13±5.43 cm, at home was 27.11±7.46 cm, the TV watching distance in school was 276.91±58.29 cm and at home was 198.61±71.34 cm. No significant correlation was found between them.

Relation between vitreous length and working time

The total writing time (school and home) were 389.89±189.69 min/wk, the total reading time (school and home) was 203.60±164.60 min/wk, the total TV time (school and home) was 831.06±536.18 min/wk. No significant correlation was found between vitreous length and working time where p > 0.05.

Relation between vitreous length and sum of writing & reading time

The writing and reading time (school and home) was 593.49±306.34 min/wk. There was no significant correlation between VL and reading and writing time where r = 0.1104 and p = 0.09.

Relation between vitreous length and working distance

The writing distance in school was 22.89±7.61 cm, at home was 25.00±7.89 cm, the reading distance in school was 25.13±5.43 cm, at home was 25.00±7.89 cm, the TV distance in school was 276.91±58.29 cm and at home was 198.61±71.34 cm. There was no significant correlation between vitreous length and reading distance where $p > 0.05$.

Diopter-hours were calculated as the sum of near work in school, home and exposure to TV (time x 1/distance) hours per week. The summation represents the measurement of child's visual activities according to the amount of accommodation in diopters (D) required to perform them. According to (Zadnik et al., 1994) formula {3 x (writing time + reading time) + 2 x (TV game time) + TV time} controlling the age, no relation was found between diopters-hours, children's axial and vitreous length where $p > 0.05$. Author used the actual reported data, in contrast to Zadnik's formula where the fixed distance have been used.

Relation between diopter-hours and axial length

The analysis shows the diopter-hours was 30.37±15.52 hours (range 7-100 hours). No significant correlation was found between axial length and diopter-hours where r = 0.0729, P = 0.26.

Relation between diopter-hours and vitreous length

The analysis shows the diopter-hours was 30.37±15.52 hours (range 7-100 hours). No significant correlation was found between vitreous length and diopter-hours where r = 0.0721, P = 0.26.

No significant relationship was found between diopter-hours and axial length ($t = 0.338$, $p = 0.736$), and between diopter hours and vitreous length ($t = 0.339$, $p = 0.735$) after controlling the age. However axial and vitreous length were found to be increased with age ($t = 3.475$, $p = 0.001$ and $t = 3.308$, $p = 0.001$ respectively). Diopter-hours and all model adjusted for age was calculated both in school and at home.

6.1.7 RELATION BETWEEN OCULAR COMPONENTS AND REFRACTION TO PARENTAL HISTORY OF MYOPIA AND EDUCATION

Two hundred and thirty seven pairs of parents gave their history of refraction and grade of education through a questionnaire. Neither parent was myopic in 120 cases (50.63%), one parent was myopic in 78 (32.91%) and both parents were myopic in 39 (16.46%).

In myopic parents group 0 (neither) the children's axial length was 21.98±0.80 mm (range 20.20-23.99 mm), group 1 (either) was 22.01±0.80 mm (range 19.67-23.89 mm) and group 2 (both) was 21.86±0.70 mm (range 20.10-23.48 mm). Distribution has been shown in Table 30. Figure 9 shows the distribution.

In myopic parents group 0 (neither) the children's vitreous length was 14.98±0.71 mm (range 13.47-17.99 mm), group 1 (either) was 15.03±0.67 mm (range 13.44-17.32 mm) and group 2 (both) was 14.91±0.51 mm (range 13.97-16.16 mm). Table 31 describes the distribution. Figure 10 shows the relation between them.

The children's refraction in parents group 0 (neither) was 0.93±0.90 D (range -1.25 to +5.00 D), group 1 (either) was 0.77±0.74 D (range -1.50 to +3.00 D) and group 2 (both) was 0.82±0.86 D (range -1.25 to +4.25 D). The distribution is shown in Figure 11. Table 27 and 35 show the parental myopia and SPEQ of children.

Using Multivariate Analysis of variance, the means of axial length, vitreous body length and refraction were found to have no significant difference for different parental myopic groups (Wilks Lambda = 0.985, p = 0.741).

Relation between maternal education and children's refraction

The extent of maternal education was grade 1= 99, grade 2 = 136 and grade 5 = 2. The mean±SD of SPEQ of mother's education of grade 1 was 0.87±0.73 D, grade 2 was 0.84±0.92 D and grade 5 was 1.62±0.18 D. No significant correlation was found between them where p = 0.43. Figure 12 shows the distribution between them. Table 28 describes the mother's education and SPEQ of children.

Relation between paternal education and children's refraction

The number of paternal education was grade 1= 75, grade 2 = 154, grade 3 = 3, grade 4 = 1 and grade 5 = 4. The Mean±SD of father's education of grade 1 was 0.94±0.78 D, grade 2 was 0.82±0.87 D, grade 3 was 1.00±0.25, grade 4 was 1.25 and grade 5 was 0.62±1.31 D. No significant correlation was found between them where p = 0.79. Distribution has been shown in Figure 13. Table 29 describes the father's education and SPEQ of children.

Table 47 & 48 represent ocular components and refractive error data on the entire sample. The estimate of the proportion of the variance explained by the models (SPEQ, CC, ACD, LT, VL, AL & LP) and controlling for 3 variables (age, diopter-hours and parental history) as a function of parental myopia and of visual task as measured by diopter-hours. Since in these method no R^2 were available, the Wilks Lambda test has been used to summarize all the models. These results show the axial and vitreous length increase with age. The results also show that children with myopic parents are more hyperopic with no significant change in optical components. Effects of parental myopia, diopter-hours and age on axial length and vitreous length were studied by multivariate analysis of variance. Parental myopia was found to have no significant effect on axial length (F = 0.3243, p = 0.723) and vitreous length (F = 0.2460, p = 0.782).

When studying the relative risk of a child with myopic parents or non-myopic parents developing myopia, odds ratios (OR) were calculated. Odds is the number of myopic children divided by the number of non-myopic children. Odds ratio were calculated among the cases with myopic parents divided by the odds among the cases with non-myopic parent. The mean SPEQ of the parents and their children were calculated. The risk of myopia for children when one of the parents is myopic was studied in terms of odds ratios (OR). Distribution has been shown in Table 32.

The prevalence of myopia was 4.22% (the relative prevalence of myopia for girls and boys according to age have been shown in Table 33). Myopia among students whose parents were both myopic only 2 (20%), either myopic 3 (30%) and whose parents were non myopic 5 (50%). This is described in Table 35. The analysis shows that the children with either or both myopic parents have no significant influence on refraction and ocular development on children (Table 38-40). The Spherical equivalent has a significant correlation with axial and vitreous length but there was no statistically significant relation with visual task. A summary of mean, range, SD, r and p value of different variables have been shown in Table 36-37.

CHAPTER 7

DISCUSSION

Neonates are hyperopic and in the majority of cases become emmetropic as they grow and develop. Congenital myopia may leads to myopic state at the pre-school children period. In these groups of subjects parental history of myopia may be a contributory factor. The pre-school children who participate in intensive visual tasks may develop myopia as a result. The eyes that will eventually become myopic may be normal in size at this time but may grow rapidly or there may be failure to compensate between refractive optics and axial elongation. The causative factors in myopia are still controversial about. Multiple investigators have used different definitions of myopia and tested subjects at different ages and different investigational methods. This makes comparisons between studies difficult in many cases.

It has been recorded (Edwards, 1991) that 95% of all children aged 3 to 6 years in Hong Kong attend a kindergarten. The kindergarten population therefore closely approximates the general population of the same age. Children in these kindergarten classes start writing English and Chinese words for many hours in school and as well as completing homework. The degree of near work effort to which these children are exposed could be a major predictive factor for the development of myopia. Hence, an early age in commencing study and long hours of near effort might have contributed to the development of myopia observed among the Chinese students (Lam & Goh, 1991). This type of study was done by Baldev et al. (1990) in India and it was found that those children who start their formal education at the age of 3-4 years are more myopic than those who start after the age of 5 years.

Chan & Edwards (1991) refracted 570 Hong Kong Chinese children aged 36 to 65 months. Both spherical and cylindrical components of the

refractive error were found to decrease with increasing age. In another investigation, Chan & Edwards (1991) measured a spherical equivalent of +0.63±0.53 D in 570 subjects having a mean age of 51.8 months. In the author's study the mean age was 59.34±10.64 months and the spherical equivalent power was +0.86±0.84 D. The prevalence of myopia was 4.22%.

Lam & Goh (1991) recorded data from 383 subjects, 144 of whom (55 girls, 89 boys) were from primary school in Hong Kong. They found that prevalence of myopia increases from 30% at age six to seven to 50% (girls) and 70% (boys) at the age 16-17. The range of spherical equivalent was between -0.01 and -1.00 D. The curve of plotted data peaks at -0.50 D and is skewed heavily towards the myopic. 40% of the children were found to be emmetropic (defined as spherical equivalent between -0.50 to +0.50 D), more than 55% were myopic (greater than -0.50 D) and less than 5% were hyperopic (greater than +0.50 D).

There was a gradual increase towards myopia with increasing age, with most of the change occurring between age 6 and 10 years.[50] Zadnik et al. (1994) observed that 12.2% of children between age 6-14 years were myopic if both parents were myopic. In contrast, only 8.2% were myopic if only one parent was myopic and only 2.7% were if neither parent was myopic. The definition of myopia was at least -0.75 D. Krause et al. (1991) found a relationship of myopic refractive error between a child's and their father's myopia but the children were measured at 20 years of age and the definition of myopia was greater than -0.25 D. In the present study there was no observed parental influence on children's eye size & refraction, although the sample size was relatively small. Sheu et al. (1983) found that only 2% of Taiwanese pre-schoolers were myopic, but follow-up study among the same group showed a prevalence of myopia of 27.3% after four years.

Refractive error strongly correlated with axial length and vitreous depth and a weaker relationship was found with anterior segment depth. Vitreous

length has the greatest influence on refraction and it is the important parameter in determining myopia since it is less disturbed during A-scan ultrasonography (Hosaka, 1988; Grosvenor, 1993). In the present study it was found that the vitreous length increases with refraction and age. Hence the vitreous length has a close relation with both refraction and age. The refractive status of the human eye is primarily determined by four factors: a) corneal curvature, b) anterior chamber depth, c) lens thickness and d) axial length. The author also found that the axial and vitreous body lengths have a close relationship with refractive status and age.

The data relating refractive status and optical components of the children were analyzed scientifically during author's study. Axial and vitreous length have a statistically significant correlation with refraction and increased age ($P < 0.05$). Several studies have found that the optical components that determine the degree of myopia were related to the elongation of vitreous chamber or the axial length (Fledelius, 1982; Lin et al, 1988; Hosaka, 1988). This study showed no correlation between the axial and vitreous lengths with amount of time devoted to visual tasks. The data suggest that increased age have a significant positive correlation with increased AL & VL and increased AL & VL have a correlation with refraction which might explain the high prevalence of myopia in this population. As myopia correlates very well with axial and vitreous length, these results suggest that increased age rather than genetic factors have a greater degree of influence in the development of myopia. No statistically significant correlation was found between refractive status and parental myopia. In addition, although parental educational level were not high, there was no correlation between this variable and children's refractive status. A large-scale study in primary school children would allow further examination of this issue, since primary school children spend more time in visual tasks than children in kindergarten. If causative factors for myopia can be identified, preventive measures and intervention therapies could possibly be devised.

Many studies have examined the development of myopia in relation to genetic and environmental influences. Several family studies have indicated a correlation between myopia in parents and offspring (Hirsch et al, 1969; Keller, 1973; Johnson et al, 1979). Studies have also demonstrated that environmental factors play an important role in the development of myopia (Anjel & Wissman, 1980; Richler & Bear, 1980; Taylor, 1982). Factors associated with myopia in Hong Kong children are unknown and may be a combination of influences. A most important developmental experience of children in Hong Kong and other Asian countries is the intensive, demanding and competitive education system. To compete effectively, students have to study at an early age and spend a great deal of time engaged in near work, which is a risk factor for development of myopia. The high academic stress in Hong Kong may be an important factor in the development of myopia at early stages of life.

Reports exist that show an increased prevalence of myopia in children with two myopic parents (Paul, 1938; Curtin, 1985). This suggests a genetic component in the etiology of myopia. However, further studies are necessary to confirm this hypothesis. Donald et al., (1993) found that if either or both parents are myopic, this increases the probability of myopia in offspring compared to knowing only refractive error at school entry or in infancy. This probability is highest if refraction at school entry is more myopic than +0.50 D and both parents are myopic. While knowing that both parents are myopic also increases the likelihood of myopia given a myopic refraction in infancy, this is less predictive of myopia than refraction at school entry. Most studies to date have examined only the genetic contribution of familial myopes to childhood myopia, while the possible hereditary effects of related hyperopes have not been studied in detail. Wold (1949) recorded a pedigree in which 10 non-myopic family members failed to alter a dominant inheritance pattern of myopia. Further work in this area, however, has not been reported. No clinical studies have been designed which can adequately distinguish the contributions

of nature or nurture to myopia. A family history of myopia is associated with the likelihood of developing myopia. A greater prevalence of myopia exists among the children of myopic parents than among the children of nonmyopic parents (Gwiazda et al., 1993; Goldschmidt, 1968; Ashton, 1985). Myopic parents with higher educational attainment may impel their children toward earlier and more rigorous scholastic activity than would non-myopic parents (Chew et al, 1988; Gawron, 1981).

Experimental and epidemiological evidence has suggested that schooling, study, reading and other visual tasks are associated with excessive axial elongation and myopia (Angel & Wissmann, 1978, 1980; Cohn, 1886; Rosner & Belkin, 1987; Ware, 1813; Young, Leary, Baldwin, West, Box, Harris & Johnson, 1969; Zylbermann, Landau & Berson, 1993). However, evidence that near work directly causes myopia is impossible to obtain from purely observational studies. Percentage of myopia increases in school children who spend number of years in school and accepted by the researchers on the basis of experiment. This finding suggests the possibility of preventing myopia by avoiding or interrupting nearwork. Richler & Bear (1980) stated that refraction is correlated with nearwork between subjects aged 5 to 60 years. It has been suggested that large amounts of nearwork in childhood may contribute to the development of myopia (Eong et al, 1993). The data presented in author's study find no correlation between refractive status and visual activities (i.e. writing, reading and TV watching).

It must be noted, however, that the subjects in our study were selected from subjects who presented for examination. Thus, we may not have seen some of the young hyperopes whose hyperopia rapidly decreased if they had no complaints and therefore no reason to seek examination. On the other hand, in many cases, we may not have seen the myopes whose myopia does not change much. These kind of studies about refraction obtained from patients of ophthalmic practice have been criticized in the past because of the selection of the population (bias of ascertainment), but Hirsch's (1952) results

from a random sample of the population were similar to those that Brown (1938) and Slataper (1950) in office patients. There was gradual increase towards myopia with increasing childhood age, with most of the change occurring between age six to ten years.

Sorsby's assertion (1970) that, if none of the grandparents nor the parents have myopia while the offspring have significant myopia, a genetic explanation is hardly tenable, is rather broad. It is true that such an occurrence excludes regular dominant inheritance, but it still leaves the possibilities of irregular dominance, recessive inheritance and polygenic inheritance. A regular dominant transmission of myopia cannot be ruled out, particularly since the mode of inheritance of myopia is not well understood and appears to involve a large number of genes (Francois, 1961).

School myopia or juvenile onset myopia is the commonest form of myopia. It usually begins between the ages eight and fourteen years with prevalence rates of about 15% while in pre-school children a rate 2% has been recorded (Zadnik, 1994). The pathogenesis of myopia is still unknown in humans, while environmental myopia has been detected as causative in animal models. Accommodation and emmetropization are two mechanisms for clarifying blurred retinal images by rapidly adjusting the optical power of the lens or slowly adjusting the length of the eye during growth (Irving et al, 1992) respectively. Insufficient accommodation may predispose to myopia and studies show that myopic children have less effective accommodation (Gwiazda et al, 1993). Because the accommodation and emmetropization behave in a parallel manner, albeit with a different time course, with more effective accommodation the retinal image will be more focused and the eye would need to elongate less in order to clear the image.

Until we know which environmental factors are important in predisposing children to myopia, it is difficult to assess the genetic factors by simply studying the similarity of children to their parents. The refractive status of children with

myopic parents would be more convincingly related to genetic influences if the differences resulted in a more myopic distribution function (Wallman J, 1994).

Richler (1980) found that nearwork had more influence on refraction at ages 5 to 14 years than at ages 60 years and up. He concluded that large amounts of nearwork in childhood might contribute to the prevalence of myopia. Myopic progression was related to time spent on reading and close work (Pärssinen & Lyyra, 1987). Levinsohn (1931) and Mohan et al. (1977) showed that the tone of extraocular muscles and hypostatic congestion in the eye caused by dependent head posture may lead to myopia in rabbit experiments. Even illumination that is used during near work may be a additional factor for causing myopia.[100]

Myopia can be predicted from consideration of early refraction determined by near retinoscopy, including both sphere and cylinder, and parent's refractive error. When children are identified at risk for myopia based on the above mentioned factors, then as ameliorative measures become better understood and they can be applied selectively to those children. Because of the limitations of human eye studies, much of our understanding of the process involved in myopia development has and will continue to come from animal research. It is important to relate this research to humans. Further work is required to develop a battery of tests which could predict the onset of juvenile myopia with both adequate sensitivity and specificity. Risk factors as predictors must be evaluated in light of the probability of the condition in the population at risk (Hill, 1987).

The growth pattern that keeps normal eyes emmetropic may be derailed in the myopic eyes. This derailment becomes more likely as the eye enlarges. Therefore, the larger an eye initially, the greater the risk of myopia (Zadnik et al., 1994). The shift in refraction in children with myopic parents would be more convincingly related to myopia if the differences were at the myopic end of the distribution. Absent the frequency distribution, the shift in average refraction could be related equally well to the inheritance (genetic) of hyperopia. Thus

even if myopia had no genetic influences, the environmental factor might have been found (Wallman J, 1994). There is still much work to be done in the important area of myopia development. Various treatments to prevent the progression of myopia have not been proven effective (Gross DA, 1982). One of the reasons is that the process and mechanisms are still obscure.[64] Author's conclusion were based on standard analytic technique. Recent research based on lower vertebrates has been designed to look exclusively at environmental causes of myopia but work in human has received little research attention since 1978. Unfortunately, no clinical studies have been designed that can adequately distinguish the contributions of nature or nurture to myopia. Eyes growing at the same rate as normal eyes would become more myopic if they are initially longer.

In China, children have to study 8 hours daily in school. They also have to do home work for at least 1 or 2 hours after school. This situation provides a heavy near point load that may relate to myopia development.[64] Myopia is the most common refractive error in Hong Kong Chinese people. The prevalence of myopia among these children increases with increasing age and exceeds 50% by the age of 10 years.[49] Children in Hong Kong are required by law to start primary school at the age of 6 or 7 years. Pupils are required to recognize and write basic Chinese characters as well as English words. They also have to undertake regular work at home. It is impossible to draw any conclusion on the relation between the start of primary school and the progression of myopia; however, the sudden increase in the amount of nearwork in primary school, when compared with that in kindergarten, may be a contributory factor. So for future study in primary school children, may revealed the causative factors of myopia.

The genetic and epidemiological background to lower degrees(less than -6 D) of refractive error is not yet well characterized.[74] Cross-sectional as well as longitudinal studies might be helpful to recognize the risk factors of myopia. Longitudinal studies of changes of refraction in association with a number of

environmental variables might well be helpful. Such studies must be stringent in the methods of selection of sample and if possible the control groups, and the definition of myopia. It is also important that to include multifactorial analysis and the appropriate statistical standardization of a number of known confounding factors. Appropriate evaluation of the nearwork-refraction relationship, and its effects on familial resemblance, will require population-based longitudinal data. It is reasonable to suspect that the extent to which effects of common familial environment are confounded with genetically determined variation in ocular refraction, differs from population to population.[80] Refraction status was determined by polygenic inheritance and considered myopia to be genetically determined (Steiger, 1913; Sorsby, 1966,1972). The influences of the environmental factors to the etiology of myopia was reported in the epidemiological and genetic surveys in Alaskan and Canadian Eskimos by Young et al (1969), Morgan & Munro (1973) and Alsbirk & Forsius (1973) discovered a low con-credence in the refraction status between the two generations. Myopia is not determined by genetic theory alone.[85] Goldschmidt (1981) described that majority of low myopia are caused by increased axial length of the eye ball. He reviewed the two theories: a) the accommodation theory and b) the convergence theory. The modernization and formal education had increased the incidence of myopia in Eskimos and Amerind population, thus indicating the contribution of environmental factors. In the India where the awareness and introduction of education at an early stage, has become a way of life and it was seen that the more younger group of people are wearing glasses.[86]

Both the heredity and environmental factors were found significantly associated with children's myopia. It seems that both parental and environment play an important role in the development of the refractive error. Parental history and other environmental factors to be make clear for better understanding of the effect of gene-environment interaction on myopia. In addition to hereditary factors, myopia could be the reason for greater axial

length, but author thinks that the studies of myopia should be included in part to determine the mechanical forces affecting the eye and sclera during close work, specially when writing and reading.[96]

Due to high density of population in a relatively confined space and the more rapid progression of myopia in the Asian, Hong Kong may be a good place to facilitate the studies. Since myopia with higher educated people have a tendency to start their children's education early therefore, it is also important to select the community of highly educated parents and family and the higher income level (M Mohan et al. 1988). For success of long term study it is important to perform all the tests in regular school hours at school grounds. Scientific data are desperately lacking about the prevalence of myopia in Hong Kong primary school children. Well-designed prospective and epidemiological studies in which several dimensions of school and home environment are comprehensively examined and provide more importance to the factors like extent of visual task, illumination, head posture, sex, education level, climate, culture, diet, ethnicity and genetics. The longitudinal type of study can confirm the hypothesis of normal eye growth in this situation. It is hopeful that this type of study will provide a epidemiological data with which community eye care programs for myopia can be planned and once determined, it can be a basis for information for a prospective study and preventive & therapeutic trials can be directed. Child's vision should be screened at least once between the ages of three and five, it should take place as early as possible, but definitely before the child enters school.[93] A long-term follow-up study of school children seems to be an interesting and rewarding one.

At the present time, it is clear that both environment and hereditary play a role in the etiology of myopia. The eye that is myopic may be normal-sized and grow faster during the onset or progression of myopia. The premyopic eye may or may not undergo normal growth patterns. Just how they do this and their relative importance still remain to be elucidated, but the foundations for understanding the etiology of myopia, particularly with the relatively recent

discovery of good animal models, are in place. As with many other things, the etiology of refractive error will probably be found to be multifactorial, with an interplay of a number of environmental factors on a genetic background. Genetic analysis does not permit assessing the importance of unknown environmental factors (Kamin, 1974), so that understanding myopia is likely to be a complicated task. Future directions of research must seek to clarify the relative contributions of numerous factors, such as stress, educational attainment, and nearwork, all of these occurring against a background of genetic influences. Such studies must be stringent in the same methods of selection of sample and same definition of myopia for comparing with other studies and there are still much work to be done in the important area of myopia development.

CHAPTER 8

REFERENCES

1. P D Trevor-Roper & P V Curran. (1984) The Eye and its Disorders. 2nd ed. chap-15, 194-198. P G Publishing Pte Ltd, Singapore.
2. Curtin Brian J. (1985) The Myopias. 3-6, 51-54, 96-97, 169-275, 177-178, 190-198. Harper & Row publishers. Philadelphia.
3. Margo., Hamed & Mames. (1994) Diagnostic Problems In Clinical Ophthalmology. 1st ed. 584-585, 587-595. W.B.Saunders Company, Philadelphia.
4. David Mittleman. (1980) Geometric Optics and Refraction. In G A Peyman., D R Sanders & M F Goldberg (ed): Principles and Practice of Ophthalmology. V-1, chap-2, 197-199. W B Saunders Company, Philadelphia.
5. Ronald C Pruett. (1994) Pathologic Myopia. In Albert & Jakobiec (ed): Principles and Practice of Ophthalmology: Clinical Practice. V-2, chap-68, 878-882. W.B. Saunders Company, Philadelphia.
6. M R Wilson & R S Baker. (1990) Epidemiology of Myopia. In proceedings of 4th International Conference of Myopia (ed): 22-27. Myopia International Research Foundation, Inc. Singapore.
7. S S Duke-Elder & David Abrams. (1970) Ophthalmic optics and Refraction. vol.5, 211 & 267-362. C.V. Mosby Company, London.
8. Blacharski P A. (1988) Pathologic progressive myopia. In Newsome D A (ed): Retinal Dystrophy and Degeneration. Chap-13, 257-269. Raven Press, New York.
9. R A Levine., M A Rosenberg & M F Rabb. (1980) The Optic Nerve. In G A Peyman., D R Sanders & M F Goldberg (ed): Principles and Practice of Ophthalmology. V-III, chap-31, 2095-2097. W B Saunders Company, Philadelphia.
10. Wang F., Li M., Zhu R & Gua X. (1990) Myopia in the Peoples Republic of China. In proceedings of 4th International Conference of Myopia (ed): 276-282. Myopia International Research Foundation, Inc. Singapore.
11. Madan M., Pakrasi S & Rajiv Z. (1988) Myopia in India. Acta Ophthalmologica (suppl). 185, 19-23.
12. Y F Shih., A H Wang & L S Ko. (1989) Refractive status of medical students in National Taiwan University. Tran of Ophth Soc of the R O C. 28, 53-58.

13. L L K Lin & L S Ko. (1988) The effect of distance gazing and eye ball exercise on the prevention of myopia progression. Acta Ophthalmologica (suppl). 185, 139-140.
14. Editorial. (1975) Changing views on myopia. British Journal of Ophthalmology. 59, 527-528.
15. Karla Zadnik., Donald O Mutti., Anthony J Adams., William A Satariona & Robert I Sholtz. (1994) Parental History and Myopia. JAMA (editorial). October 26, V-272, N-16, p1256.
16. Editorials. (1994) A New Focus on Myopia. JAMA. May 4, 271, 17, 1362- 1363.
17. Karla Zadnik., Donald O Mutti., Anthony J Adams., William A Satariona & Robert I Sholtz. (1994) The Effect of Parental History of Myopia on Children's Eye Size. JAMA. May 4, V-271, N-17, 1323-1327.
18. P Hanczyc. (1990) Anthropologic Characteristics of High Myopia patients. In proceedings of 4th International Conference of Myopia (ed): 28-45. Myopia International Research Foundation, Inc. Singapore.
19. Luke L J L., Por T H., Liang S K & Ping KH. (1988) Study of myopia among aboriginal school children in Taiwan. Acta Ophthalmologica (suppl). 185, 34-36.
20. Ruth Riise. (1988) Myopia as cause of blindness. Acta Ophthalmologica (suppl). 185, 109-110.
21. R D Sperduto., D Seigel., J Roberts & M Rowland. (1983) Prevalence of myopia in the United States. Arch Ophthalmology. 101, 405-407.
22. S J Chew., S C Chia & L K H Lee. (1988) The Pattern of Myopia in Young Singaporean Men. Singapore Medical Journal. 29, 201-211.
23. D J Apple & G O H Naumann. (1986) Malformation and Anomalies of the Eye. In Neumann and Apple (ed): Pathology of the Eye. chap-3, 76-80. Springer-Verlag, New York.
24. Steven M Poods & Myron Yanoff. (1991) Optics and Refraction. V-1, pp 8.2. Gower Medical Publishing, New York.
25. K Y Wu., M M Sheu., W L Huang., H Z Wang and C W Chen. (1990) A Longitudinal Study on the Refraction and Its Components among Children of Kaohsiung City, Taiwan. In proceedings of 4th International Conference of Myopia (ed): 254-262. Myopia International Research Foundation, Inc. Singapore.
26. M Rosner & M Belkin. (1990) Association of Myopia with Years of School Attendance and Intelligence. In proceedings of 4th International Conference of Myopia (ed): 272-275. Myopia International Research Foundation, Inc. Singapore.
27. Laatikainen L., Tolppanen & Harju H. (1985) Epidemiology of rhegmatogenous retinal detachment in a Finnish population. Acta Ophthalmologica. 63, 59-64.

28. Ragnar T., Staffan S & Per T. (1987) Retinal detachment-A study of a population based patient material in Sweden 1971-1981. Acta Ophthalmologica. 65, 213-222.
29. Josh Wallman & Julian I Adams. (1987) Developmental Aspects of Experimental Myopia in Chicks: Susceptibility, Recovery and Relation to Emmetropization. Vision Research. 27, 7, 1139-1163.
30. B P Hayes., F W Fitzke., W Hodos & A L Holden. (1986) A Morphological Analysis of Experimental Myopia in Young Chickens. Investigative Ophthalmology & Visual Science. 27, 981-991.
31. J C Lin., Y F Shih., L L K Lin & P T Hung. (1993) The Role of Environmental and Familial Factors in the Causation of High Myopia—A Questionnaire Study. Academic and Annual Meeting of the Ophthalmological Society of the Republic of China (Abstract).
32. Alec Garnee & Gordon K. (1994) Pathobiology of Ocular Diseases. Part-A, 2nd Edn. Marcel Dekker, Inc, New York.
33. Basic & Clinical Science Course. (1994-95) Section 2, 3, 6 & 11. American Academy of ophthalmology.
34. Liang-yen Wen. (1988) Myopic Chorioretinal Degeneration. In Mark O M Tso (ed): Retinal Diseases: Biomedical Foundations and Clinical Management. chap-14, 242-246. J B Lippincott Company, Philadelphia.
35. Brian Curtin. (1988) Optical components, refraction and age: various myopic types. Acta Ophthalmologica (suppl). Editorial. 185, 63-64.
36. Luke L K L., Li F H., Yung F S., Por T H & Liang S K. (1988) Correlation of optical components with ocular refraction among teen-agers in Taipei. Acta Ophthalmologica (suppl). 185, 69-73.
37. L L K Lin., T H Wang., J H Jan., Y F Shih & L S Ko. (1991) Survey of the ocular refraction with its optical components among freshmen in National Taiwan University. Tran of Ophth Soc of the R O C. 30, 597-604.
38. Akio Hosaka. (1988) Population studies- myopia experience in Japan. Acta Ophthalmologica (suppl). 185, 37-40.
39. H C Fledelius. (1988) Corneal curvature radius. Acta Ophthalmologica (suppl). 185, 75-77.
40. Karl G Nyman. (1988) Occupational near-work myopia. Acta Ophthalmologica (suppl). 185, 167-171.
41. Maija I Mäntyjärvi. (1985) Changes of Refraction in Schoolchildren. Archives of Ophthalmology. 103, 790-792.
42. W G Whitmore. (1992) Congenital and developmental myopia. Eye. 6, 361- 365.
43. H C Fledelius & M Stubgaard. (1986) Changes in refraction and corneal curvature during growth and adult life. Acta Ophthalmologica. 64, 487-491.

44. A T More. (1985) Refraction in Children. Trans Ophthalmol Soc. UK. 104, 648-652.
45. Leon F Garner., Robert F Kinner., Jeremy D Klinger and Malcolm J McKeller. (1985) Prevalence of myopia in school children in Vanuatu. Acta Ophthalmologica. 63, 323-326.
46. Health report-the good news. (1994) Time. 16th May. p 38.
47. M A Sharif & M Amanullah. (1991) Prevalence of refractive Error in Urban School-Children of Bangladesh. Trans of O S B. 18(2), 20-24.
48. Edwards M. (1991) The refractive status of Hong Kong Chinese infants. Ophthal Physiol Opt. 11, 297-303.
49. Chan OYC & Edwards M. (1993) Refractive errors in Hong Kong Chinese pre-school children. Optometry and Vision Science. 70(6), 501-505.
50. Lam CSY & Goh WSH. (1991) The incidence of refractive errors among school children in Hong Kong and its relationship with the optical components. Clinical and Experimental Optometry. 74, 97-103.
51. Rosner M & Belkin M (1987) Intelligence,Education and Myopia in males. Arch Ophthalmol. 105, 1508-1511.
52. Richler A & Bear JC. (1980) Refraction, Nearwork and Education. A population study in Newfoundland. Acta Ophthalmologica. 58, 468-478.
53. Jennifer H., Leilani P & Howard C H. (1995) Correlation Between Familial Refractive Error and Children's Non-cycloplegic Refraction. Vision Research. 35(9), 1353-1358.
54. Arnold Sorsby. (1970) Transmission of Refractive Errors within Eskimo Families. Am J of Optom Arch Am Acad Optom. 47: 244-249.
55. Donald O Mutti & Karla Zadnik. (1995) The Utility of Three Predictors of Childhood Myopia: a Bayesian Analysis. Vision Research. 35(9), 1345-1352.
56. J Gwiazda., F Thorn., J Bauer & R Held. (1993) Emmetropization and the progression of manifest refraction in children followed from infancy to puberty. Clin Vision Sci. 8(4), 337-344.
57. W G Whitmore. (1991) The etiology of myopia. Current Opinion in Ophthalmology. 2, 72-77.
58. Baldewin WR. (1981) A review of statistical studies of refraction between myopia and ethnic, behavioural and physiological characteristics. American Journal of Optometry and Physiological Optics. 58(7), 516-527.
59. Lin LLK., Chen CJ., Hung PT & Ko LS. (1988) Nation-wide survey of myopia among school-children in Taiwan, 1986. Acta Ophthalmologica (suppl). 185, 29-33.

60. David A.Goss & Paul Erickson. (1987) Meridional Corneal Components of Myopia Progression in Young Adults and Children. Am J of Optometry & Physiological Optics. 64(7), 475-481.
61. Maija I. Märtyjärvi. (1985) Changes of Refraction in Schoolchildren. Arch Ophthalmol. 103, 790-792.
62. Robert A Gordon & Paul B Donzis. (1985) Refractive Development of the Human Eye. Arch Ophthalmol. 103, 785-789.
63. Theodore Grosvenor. (1970) Refractive error distribution in New Zealand's Polynesian And European Children. Am J Optom Arch Am Acad Optom. 47: 673-679.
64. Q Mei & Z Rong. (1994) Early Signs of Myopia in Chinese Schoolchildren. Optometry and Vision science. 71 (1): 14-16.
65. M Edwards & OYC Chan. (1994) Refraction referral criteria for Hong Kong Chinese pre-school children. Ophthal. Physiol. Opt. 14, 249-256.
66. Yap M & Edwards M. (1990) Visual problems in Hong Kong primary school children. Clinical & Experimental Optometry. 73, 58-63.
67. M.Yap., M. Wu., Z.M.Liu., F L Lee & H. Wang. (1993) Role of heredity in the genesis of myopia. Ophthal. Physiol. Opt. 13, 316-319.
68. Marion Edwards. (1991) The refractive status of Hong Kong Chinese infants. Ophthal. Physiol. Opt. 11, 303.
69. Graham E Q., Jesse A B., Terri L Y., Sule Z & Richard A S. (1995) Association of Intraocular Pressure and Myopia in Children. Ophthalmology. 102, 180-185.
70. Marion H. Edwards & Brian Brown. (1993) Intraocular Pressure in a Selected Sample of Myopic and Nonmyopic Chinese Children. Optometry and Vision Science. 70(1), 15-17.
71. Jennifer LW & William H. (1991) Intraocular pressure and eye enlargement in chicks. Current Eye Research. 10(2), 163-168.
72. Ulf HK., Paula TR., Markku JK & Jyrki KM. (1992) The Development of Myopia up to the Age of Twenty and a Comparison of Refraction in Parents and Children. Arctic Medical Research. 52, 161-165.
73. J Angel & D A Wissmann. (1980) The Epidemiology of Myopia. American Journal of Epidemiology. 111(2), 220-228.
74. J C Bear & A Richler. (1982) Environmental Influences on Ocular Refraction (Letters to the editor). American Journal of Epidemiology. 115(1), 138-142.
75. John Angle & D A Wissmann. (1978) Age, Reading and Myopia. Am J of Optom & Physiol. Opt. 55(5), 302-308.

76. Geoffrey C A. (1985) Nearwork, School Achievement and Myopia. Journal of Biosocial Science. 17, 223-233.

77. M S Banks. (1980) Infant Refraction and Accommodation. International Ophthalmology. 20, 205-232.

78. M J Hirsch & F W Weymouth. (1990) Changes in Optical Components. In T Grosvenor & M C Flom (ed): Refractive Anomalies: Research and Clinical Application. chap-3, 39-56. Butterworth-Heinemann, Boston.

79. I Mohindra & R Held. (1981) Refraction in human from birth to five years. In H C Fledelius., P H Alsbirk & E Goldschmidt (ed): Documenta Ophthalmologica Proceedings Series: Third International Conference on Myopia,1981. 28, 19-27. Dr. W Junk Publishers, The Hague.

80. A Richler & J C Bear. (1981) Nearwork and familial resemblance in ocular refraction: A population study in Newfoundland. In H C Fledelius., P H Alsbirk & E Goldschmidt (ed): Documenta Ophthalmologica Proceedings Series: Third International Conference on Myopia,1981. 28, 41-46. Dr. W Junk Publishers, The Hague.

81. H Gernet (1981) Oculometric Findings in Myopia. In H C Fledelius., P H Alsbirk & E Goldschmidt (ed): Documenta Ophthalmologica Proceedings Series: Third International Conference on Myopia,1981. 28, 71-77. Dr. W Junk Publishers, The Hague.

82. H C Fledelius. (1981) Refractive components in aniso and isometropia. An oculometric study (by ultrasonography and keratometry). In H C Fledelius., P H Alsbirk & E Goldschmidt (ed): Documenta Ophthalmologica Proceedings Series: Third International Conference on Myopia,1981. 28, 89-95. Dr. W Junk Publishers, The Hague.

83. U Rajan., F T Tan., T K Chan., J W Dvorak & S J Chew. (1994) Changing Prevalence of Myopia in Singapore School Children. In proceedings of 5th International Conference of Myopia (ed): 41-48. Myopia International Research Foundation, Inc. New York-Singapore.

84. M Mäntyjärvi. (1990) High Myopia in a Family. In proceedings of 4th International Conference of Myopia (ed): 89-97. Myopia International Research Foundation, Inc. Singapore.

85. S Y Lin., L L K Lin, J H Jan & C J Chen. (1990) Family Study on Ocular Refraction. In proceedings of 4th International Conference of Myopia (ed): 151-159. Myopia International Research Foundation, Inc. Singapore.

86. G Baldev., D Mangat., S Balram., C Kulbhusan., P Ranjit & P Raman. (1990) Myopia in Relation to Early Schooling. In proceedings of 4th International Conference of Myopia (ed): 283-293. Myopia International Research Foundation, Inc. Singapore.

87. O Pärssinen. (1990) Eye movements as a link between reading and myopic progression. In proceedings of 4th International Conference of Myopia (ed): 342-353. Myopia International Research Foundation, Inc. Singapore.

88. Takasi Sato. (1986) The relationship between the growth of the ocular axis and the crystalline lens in the case of school myopia. In proceedings of 3rd International Conference of Myopia (ed): 56-76. Myopia International Research Foundation, Inc. Rome.

89. M M Sheu., W L Huang., C W Chen & H Z Wang. (1986) A study of refraction in kindergarten children in Kaohsiung. In proceedings of 3rd International Conference of Myopia (ed): 267-284. Myopia International Research Foundation, Inc. Rome.

90. David Troilo. (1992) Neonatal Eye Growth and Emmetropisation-A Literature Review. Eye. 6, 154-160.

91. M J Hirsch & D L Ditmars. (1969) Refraction of young myopes and their parents-A reanalyses. Am J of Optom and Arch of Am Acad of Optom. 46(1), 30-32.

92. C J Chen., B H Cohen & E L Diamond. (1985) Genetic and environmental effects on the development of myopia in Chinese twin children. Ophthalmic Paediatrics and Genetics. 6(1/2), 113-119.

93. Elizabeth M H. (1979) Methods and Standards For Screening Pre-school Children. The Sightsaving Review. 49, 71-83.

94. P A Blacharski. (1988) Pathologic Progressive Myopia. In David A. Newsome (ed): Retinal Dystrophies and Degeneration. chap-13, 257-269. Raven Press, New York.

95. J J Kanski. (1994) Principles of refractive corneal surgery. chap-5, 143-145. Butterworth-Heinemann Ltd, 1994, Reprinted 1995.

96. O Pärssinen & A L Lyyra. (1993) Myopia and Myopic Progression Among Schoolchildren: A Three-Year Follow-Up Study. Investigative Ophthalmology & Visual Science. 34 (9), 2794-2802.

97. L F Garner., R F Kinnear., M McKellar., J Klinger & M S Hovander. (1988) Refraction and Its Components in Melanesian Schoolchildren in Vanuatu. Am J of Optom & Physio Optics. 65 (3), 182-189.

98. J P Leung., J S M Lai., W C W Hsu & P C P Ho. (1987) Generalisation of the effects of behavioral training for myopia. Behavior and Research Therapy. 25(2), 159-163.

99. L L K Lin & C J Chen. (1988) A twin study in Chinese school children. Acta Ophthalmologica. 185 (suppl), 51-53.

100. M Mohan., S Pakrasi & S P Garg. (1988) The role of environmental factors and hereditary predisposition in the causation of low myopia. Acta Ophthalmologica. 185 (suppl), 55-57.

101. Teikari JM., O'Donnell J & Koskenvuo. (1991) Impact of heredity in myopia. Human Heredity. 41(3), 151-156.

102. Francis A Young & G A Leary. (1972) The Inheritance of Ocular Components. Am J of Optom and Arch of Am Acad of Optommetry. 546-555.

TABLES

Table 1(a). DISTRIBUTION OF REFRACTION(right eye)

SPEQ(D)	Frequency	Percent(%)
< -0.50	10	4.2
-0.50 to +0.50	68	28.7
> +0.50	159	67.1

Table 1(b). DISTRIBUTION OF REFRACTION(left eye)

SPEQ(D)	Frequency	Percent(%)
< -0.50	16	6.8
-0.50 to +0.50	74	31.2
> +0.50	147	62.0

Table 2. PERFORMANCE OF VISUAL ACUITY(PH)

Vision	Frequency	Percent(%)	Girls	Boys
1.00(20/20)	207	87.34	105	102
0.67(20/30)	30	12.66	9	21

Table 3. AGE DISTRIBUTION

Age(month)	Frequency	Percent(%)
36-47	42	17.72
48-59	76	32.07
60-71	78	32.91
72-83	41	17.30

Table 4. AXIAL LENGTH DISTRIBUTION

AL(mm)	Frequency	Percent(%)
<20	1	0.42
20-20.99	25	10.55
21-21.99	99	41.77
22-22.99	90	37.98
≥23	22	9.28

Table 5. DISTRIBUTION OF VITREOUS BODY LENGTH

VL(mm)	Frequency	Percent(%)
13-13.99	16	6.75
14-14.99	103	43.46
15-15.99	106	44.73
≥16	12	5.06

Table 6. DISTRIBUTION OF INTRAOCULAR PRESSURE

IOP(mm Hg)	Frequency	Percent(%)
<10	11	4.64
10-21	224	94.51
>21	2	0.84

Table 7. DIFFERENCE IN REFRACTIVE ERROR BETWEEN SEXES USING SEPARATE-VARIANCE TEST(expressed in D)

Sex	No.of Subj	Mean Sphere (SD)	Mean Cylinder (SD)	Mean Spherical Equiv.(SD)
Girls	114	1.22(0.81)	-0.64(0.48)	0.94(0.77)
Boys	123	1.10(0.90)	-0.64(0.48)	0.77(0.90)

Table 8. CHILDREN GROUPED ON THE BASIS OF SEX AND REFRACTIVE ERROR

Refractive Error	Boys(%)	Girls(%)	Total	Percent(%)
Myopic	8(3.4)	2(0.84)	10	4.22
Emmetropic	38(16.0)	30(12.7)	68	28.69
Hyperopic	77(32.5)	82(34.6)	159	67.09

Table 9. SPHERICAL EQUIVALENT WITH AGE DISTRIBUTION

Group(mth)	No	Mean(D)	SD	SE	95% CI
36-47	42	0.84	0.81	0.12	0.58-1.09
48-59	76	0.96	1.00	0.12	0.73-1.19
60-71	78	0.81	0.81	0.09	0.63-0.99
72-83	41	0.78	0.62	0.10	0.58-0.97
TOTAL	237	0.86	0.84	0.05	0.75-0.97

Table 10. AXIAL LENGTH WITH AGE DISTRIBUTION

Group(mth)	No	Mean(mm)	SD	SE	95% CI
36-47	42	21.63	0.78	0.12	21.38-21.87
48-59	76	21.92	0.70	0.08	21.76-22.08
60-71	78	22.13	0.84	0.09	21.94-22.31
72-83	41	22.11	0.74	0.11	21.88-22.34
TOTAL	237	21.97	0.79	0.05	21.87-22.07

Table 11. VITREOUS LENGTH WITH AGE DISTRIBUTION

Group(mth)	No	Mean(mm)	SD	SE	95% CI
36-47	42	14.75	0.54	0.08	14.58-14.92
48-59	76	14.93	0.63	0.07	14.79-15.08
60-71	78	15.10	0.76	0.08	14.93-15.27
72-83	41	15.12	0.60	0.09	14.93-15.31
TOTAL	237	14.99	0.67	0.04	14.90-15.08

Table 12. DISTRIBUTION OF SCHOOL WRITING (distance)

Distance(cm)	Frequency	Percent(%)
≤15	29	12.24
16-20	74	31.22
21-25	77	32.49
26-30	32	13.50
>30	25	10.55

Table 13. DISTRIBUTION OF SCHOOL READING (distance)

Distance(cm)	Frequency	Percent(%)
≤15	1	0.42
16-20	33	13.92
21-25	118	49.79
26-30	58	24.48
>30	27	11.39

Table 14. DISTRIBUTION OF SCHOOL TV WATCHING(distance)

Distance(cm)	Frequency	Percent(%)
150-200	37	15.61
201-250	39	16.46
251-300	92	38.82
301-350	42	17.72
≥350	27	11.39

Table 15. DISTRIBUTION OF SCHOOL WRITING (time)

Time(minute)	Frequency	Percent(%)
≤60	9	3.80
61-120	144	60.76
121-180	82	34.60
≥180	2	0.84

Table 16. DISTRIBUTION OF SCHOOL READING (time)

Time(minute)	Frequency	Percent(%)
≤60	174	73.42
>60	63	26.58

Table 17. DISTRIBUTION OF SCHOOL TV WATCHING(time)

Time(minute)	Frequency	Percent(%)
30≤	198	83.54
>30	39	16.46

Table 18. DISTRIBUTION OF HOME WRITING (distance)

Distance(cm)	Frequency	Percent(%)
<16	18	7.59
16-20	70	29.54
21-25	66	27.85
26-30	38	16.03
>30	45	18.99

Table 19. DISTRIBUTION OF HOME READING (distance)

Distance(cm)	Frequency	Percent(%)
<16	8	3.37
16-20	49	20.67
21-25	63	26.58
26-30	62	26.16
>30	55	23.21

Table 20. DISTRIBUTION OF HOME TV WATCHING (distance)

Distance(cm)	Frequency	Percent(%)
≤150	74	55.70
151-200	56	23.63
201-300	94	39.66
>300	13	5.48

Table 21. DISTRIBUTION OF HOME WRITING (time)

Time(minute)	Frequency	Percent(%)
≤120	36	15.19
121-240	98	41.35
241-360	52	21.94
361-480	28	11.81
481-600	6	2.53
601-720	9	3.80
721-840	5	2.11
841-960	2	0.84
>960	1	0.42

Table 22. DISTRIBUTION OF HOME READING (time)

Time(minute)	Frequency	Percent(%)
0	22	9.28
10-120	113	47.70
121-240	62	26.16
241-360	22	9.28
361-480	9	3.80
481-600	3	1.26
601-720	2	0.84
>720	4	1.68

Table 23. DISTRIBUTION OF HOME TV WATCHING(time)

Time(minute)	Frequency	Percent(%)
0	7	2.95
30-360	46	19.41
361-720	57	24.05
721-1080	70	29.53
1081-1440	28	11.81
1441-1800	13	5.48
1801-2160	11	4.64
2161-2520	3	1.26
2521-3020	2	0.84

Table 24. DISTRIBUTION OF SCHOOL DIOPTER-HOUR

DH (hr/wk)	Frequency	Percent(%)
8≤	125	52.74
>8	112	47.26

Table 25. DISTRIBUTION OF HOME DIOPTER-HOUR

DH(hr/wk)	Frequency	Percent(%)
24≤	81	34.17
25-47	112	47.26
48-71	30	12.66
≥72	14	5.91

Table 26. DISTRIBUTION OF SCHOOL & HOME DIOPTER-HOUR

DH(hr/wk)	Frequency	Percent(%)
24≤	31	13.08
25-47	125	52.74
48-71	60	25.32
≥72	21	8.86

Table 27. DISTRIBUTION OF PARENTAL MYOPIA AND SPEQ OF CHILDREN

Group 0=neither, 1=either and 2=both are myopic

Group	No	Mean(D)	SD	SE	95% CI
0	120	0.93	0.90	0.08	0.76-1.09
1	78	0.77	0.74	0.08	0.60-0.94
2	39	0.82	0.86	0.14	0.54-1.10
TOTAL	237	0.86	0.84	0.05	0.75-0.97

Table 28. DISTRIBUTION OF MOTHER'S EDUCATION AND SPEQ OF CHILDREN

Grade	No	Mean(D)	SD	SE	95% CI
1	99	0.87	0.73	0.07	0.72-1.01
2	136	0.84	0.92	0.08	0.68-1.00
5	2	1.62	0.18	0.12	0.04-3.21
TOTAL	237	0.86	0.84	0.05	0.75-0.97

* 3 & 4 grade were absent

Table 29. DISTRIBUTION OF FATHER'S EDUCATION AND SPEQ OF CHILDREN

Grade	No	Mean(D)	SD	SE	95% CI
1	75	0.94	0.78	0.09	0.76-1.12
2	154	0.82	0.87	0.07	0.68-0.96
3	3	1.00	0.25	0.14	0.38-1.62
4*	1	1.25			
5	4	0.62	1.31	0.66	-1.46-2.72
TOTAL	237	0.86	0.84	0.05	0.75-0.97

* This age group was too small

Table 30. DISTRIBUTION OF PARENTAL MYOPIA AND AXIAL LENGTH OF CHILDREN

Group 0=neither, 1=either and 2=both are myopic

Group	No	Mean(D)	SD	SE	95% CI
0	120	21.98	0.80	0.07	21.84-22.13
1	78	22.01	0.80	0.09	21.83-22.19
2	39	21.86	0.70	0.11	21.64-22.09
TOTAL	237	21.97	0.79	0.05	21.87-22.07

Table 31. DISTRIBUTION OF PARENTAL MYOPIA AND VITREOUS LENGTH OF CHILDREN

Group 0=neither, 1=either and 2=both are myopic

Group	No	Mean(D)	SD	SE	95% CI
0	120	14.99	0.71	0.06	14.86-15.11
1	78	15.03	0.67	0.08	14.88-15.19
2	39	14.92	0.51	0.08	14.75-15.08
TOTAL	237	14.99	0.67	0.04	14.90-15.08

Table 32. ESTIMATES OF ODDS RATIO OF MYOPIA AMONG CHILDREN ACCORDING TO MYOPIA IN THEIR PARENTS. OR = ODDS RATIO, 95% CI = 95% CONFIDENCE INTERVAL

Factor	Girls (OR)	95% CI	Boys(OR)	95% CI
Mother	2.03	0.12~33.32	0.90	0.20~3.95
Father	2.50	0.15~41.19	0.79	0.15~4.13

Table 33. RELATIVE PREVALENCE OF MYOPIA FOR GIRLS AND BOYS ACCORDING TO AGE

Age(yr.)	Sex	Percent myopic(%)	Sex	Percent myopic(%)	Ratio	Total(M/F)
3	M	2(20.0)	F	1(10.0)	2:1	24/18
4	M	2(20.0)	F	1(10.0)	2:1	41/33
5	M	4(40.0)	F*	0		43/36
6*	M	0	F	0		15/27

* This age group was non myopic

Table 34. AGE DISTRIBUTION WITHIN THE MYOPIC AND NON-MYOPIC GROUPS

Age(yr.)	Myopic Group (N)	Non-myopic Group (N)
3	3	39
4	3	71
5	4	75
6*		42

* This group has no myopia

Table 35. PRESENCE OF MYOPIA AMONG PARENTS OF MYOPIC CHILDREN

Parent (%)	Boys N(%)	Girls N(%)	Total N(%)
Both Parents Myopic(16.46)	1(10.0)	1(10.0)	2(20.0)
One Parent Myopic(32.91)	3(30.0)	*	3(30.0)
Neither Parent Myopic(50.63)	4(40.0)	1(10.0)	5(50.0)

* This group has no myopia

Table 36. SUMMARY OF MEAN, RANGE & SD

FACTORS	MEAN	RANGE	SD
Age(month)	59.34	36-83	10.64
Spherical Equivalent(D)	0.86	-1.5 ~+5	0.84
Axial length(mm)	21.97	19.67-23.99	0.79
Vitreous length(mm)	14.99	13.44-17.99	0.67
IOP(mm Hg)	14.54	6-21.7	2.30
Writing time(min/wk)	389.89	60-1385	189.69
Reading time(min/wk)	203.60	20-1165	164.60
TV time(min/wk)	831.06	10-3060	536.18
S-writ-distance(cm)	22.89	10-40	7.61
S-read-distance(cm)	25.13	15-58	5.43
H-read-distance(cm)	27.11	10-53	7.46
H-writ-distance(cm)	25.00	10-50	7.89
S-TV-distance(cm)	276.91	175-380	58.29
H-TV-distance(cm)	198.61	30-459	71.34
Diopter-hours(hr/wk)	30.37	7-100	15.52

Table 37. SUMMARY OF CORRELATION COEFFICIENT(r) & P VALUE

RELATIONS	r	p
Age & axial length	0.2347	<0.001
Age & vitreous length	0.2245	<0.001
SPEQ & axial length	-0.4396	<0.001
SPEQ & vitreous length	-0.3942	<0.001
Parent & axial length		0.63
Parent & vitreous length		0.67
Parent & SPEQ		0.42
Mother's education & SPEQ		0.43
Father's education & SPEQ		0.79
Diopter-hours & SPEQ	-0.0166	0.80
Diopter-hours & axial length	0.0729	>0.05
Diopter-hours & vitreous length	0.0721	>0.05
Visual task & SPEQ		>0.05

Table 38. COMPARISON BETWEEN PARENTS AND CHILDREN

Parent (%)	SPEQ	AL	VL
Both Parents Myopic(16.46)	-1.25~4.25	20.10-23.48	13.97-16.16
One Parent Myopic(32.91)	-1.5~3.00	19.67-23.89	13.44-17.32
Neither Parent Myopic(50.63)	-1.25~5.00	20.20-23.99	13.47-17.99

Table 39. DISTRIBUTION OF FATHER'S EDUCATION AND SPEQ(D) OF CHILDREN

Grade	No	SPEQ(mean)	MINIMUM	MAXIMUM
1	75	0.94	-1.00	+3.00
2	154	0.82	-1.5	+5.00
3	3	1.00	+0.75	+1.25
4*	1	1.25	+1.25	+1.25
5	4	0.62	-0.75	+1.75
TOTAL	237	0.86	-1.5	+5.00

* This age group was too small

Table 40. DISTRIBUTION OF MOTHER'S EDUCATION AND SPEQ(D) OF CHILDREN

Grade	No	SPEQ(mean)	MINIMUM	MAXIMUM
1	99	0.87	-1.25	+3.00
2	136	0.84	-1.5	+5.00
5	2	1.62	-1.25	+3.00
TOTAL	237	0.86	-1.5	+5.00

* 3 & 4 grade were absent

Table 41. AGE DISTRIBUTION OF SPEQ AND OPTICAL COMPONENTS(mean in mm) AND IOP(mean)

Age(yr)	SPEQ(D)	AL	VL	IOP(mmHg)
3	-1.25~3	21.63	14.75	13.87
4	-1.5~5	21.92	14.94	14.52
5	-1.25~4	22.13	15.10	14.60
6	-0.25~2.75	22.11	15.12	15.12

Table 42. RELATION (grouping)BETWEEN DIOPTER-HOUR AND AL(mm) & VL(mm)

DH(hr/wk)	AL(mean)	VL(mean)
24≤	21.81	14.86
25-47	22.07	15.10
48-71	22.11	15.18
≥72	21.81	14.70

Table 43. RELATION WITH AGE DISTRIBUTION & DIOPTER-HOURS (grouping)

Group(mth)	No	Mean(hr)	RANGE
36-47	42	27.63	7-84
48-59	76	24.18	9-64
60-71	78	36.41	12-100
72-83	41	32.61	13-100
TOTAL	237	30.37	7-100

Table 44. DISTRIBUTION OF SCHOOL WRITING (distance) & AXIAL LENGTH (grouping in mean)

Distance(cm)	AL(mm)	RANGE
15≤	21.86	20.26-23.05
16-30	21.93	19.67-23.89
>30	22.36	21.46-23.99

Table 45. DISTRIBUTION OF SCHOOL TV WATCHING(distance) & AXIAL LENGTH (grouping in mean)

Distance(cm)	AL (mm)	RANGE
180≤	21.66	20.20-23.09
181-359	22.03	19.67-23.99
≥360	22.04	23.01-21.79

Table 46. DISTRIBUTION OF SCHOOL TV WATCHING(distance) & VITREOUS LENGTH (grouping in mean)

Distance(cm)	VL (mm)	RANGE
180≤	14.70	13.47-16.28
181-359	15.05	13.44-17.99
≥360	14.97	14.00-15.85

Table 47. ADJUSTED MEANS OF OCULAR COMPONENTS BY PARENTAL HISTORY(Controlling for age in month and Diopter-hours)

Parent Myopic(N)	SPEQ(D)	CC(D)	ACD(mm)	LT(mm)	AL(mm)	VL(mm)	LP(D)	AL+
Neither(N=120)	0.927	44.27	3.18	3.81	21.98	14.99	22.96	N.A.
Either(N=78)	0.772	44.01	3.20	3.77	21.99	15.02	23.40	N.A.
Both(N=39)	0.818	44.20	3.15	3.77	21.88	14.93	23.49	N.A.
Wilks Lambda	0.961# (for all model)							
P-value for								
Parent History	0.435	0.494	0.827	0.712	0.723	0.782	0.314	N.A.
Age in month	0.589	0.828	0.365	0.564	0.001*	0.001*	0.005*	0.001*
Diopter-Hours	0.968	0.223	0.435	0.480	0.736	0.735	0.345	0.736

Remarks: *- Axial length, vitreous length and lens power increase with age.
+- Model for axial length without parent myopia.
#- In these model no R^2 were available, so Wilks Lambda has been used to summarize.

Table 48. ADJUSTED MEANS OF OCULAR COMPONENTS BY PARENTAL HISTORY(Controlling for Diopter-hours only)

Parent Myopic(N)	SPEQ(D)	CC(D)	ACD(mm)	LT(mm)	AL(mm)	VL(mm)	LP(D)	AL+
Neither(N=120)	0.926	44.27	3.18	3.81	21.98	14.99	22.95	N.A.
Either(N=78)	0.770	44.01	3.20	3.78	22.00	15.03	23.37	N.A.
Both(N=39)	0.820	44.17	3.15	3.77	21.86	14.92	23.52	N.A.
Wilks Lambda	0.959# (for all model)							
P-value for								
Parent History	0.431	0.490	0.803	0.708	0.639	0.699	0.312	N.A.
Diopter-Hours	0.870	0.229	0.551	0.393	0.270	0.285	0.115	0.270

Remarks: +- Model for axial length without parent myopia.
#- In these model no R^2 were available, so Wilks Lambda has been used to summarize.

Table 49. DISTRIBUTION OF IOP(mm Hg) & AXIAL LENGTH (grouping in mean)

IOP	AL (mm)	SD	RANGE
<10	22.00	0.57	21.13-23.05
10-21	21.97	0.80	19.67-23.99
>20	22.14	0.35	21.90-22.39

Table 50. DISTRIBUTION OF IOP(mm Hg) & VITREOUS LENGTH (grouping in mean)

IOP	VL (mm)	SD	RANGE
<10	15.00	0.58	14.06-16.32
10-21	14.99	0.67	13.44-17.99
>20	14.91	0.24	14.74-15.08

Figure 1. Distribution of Age

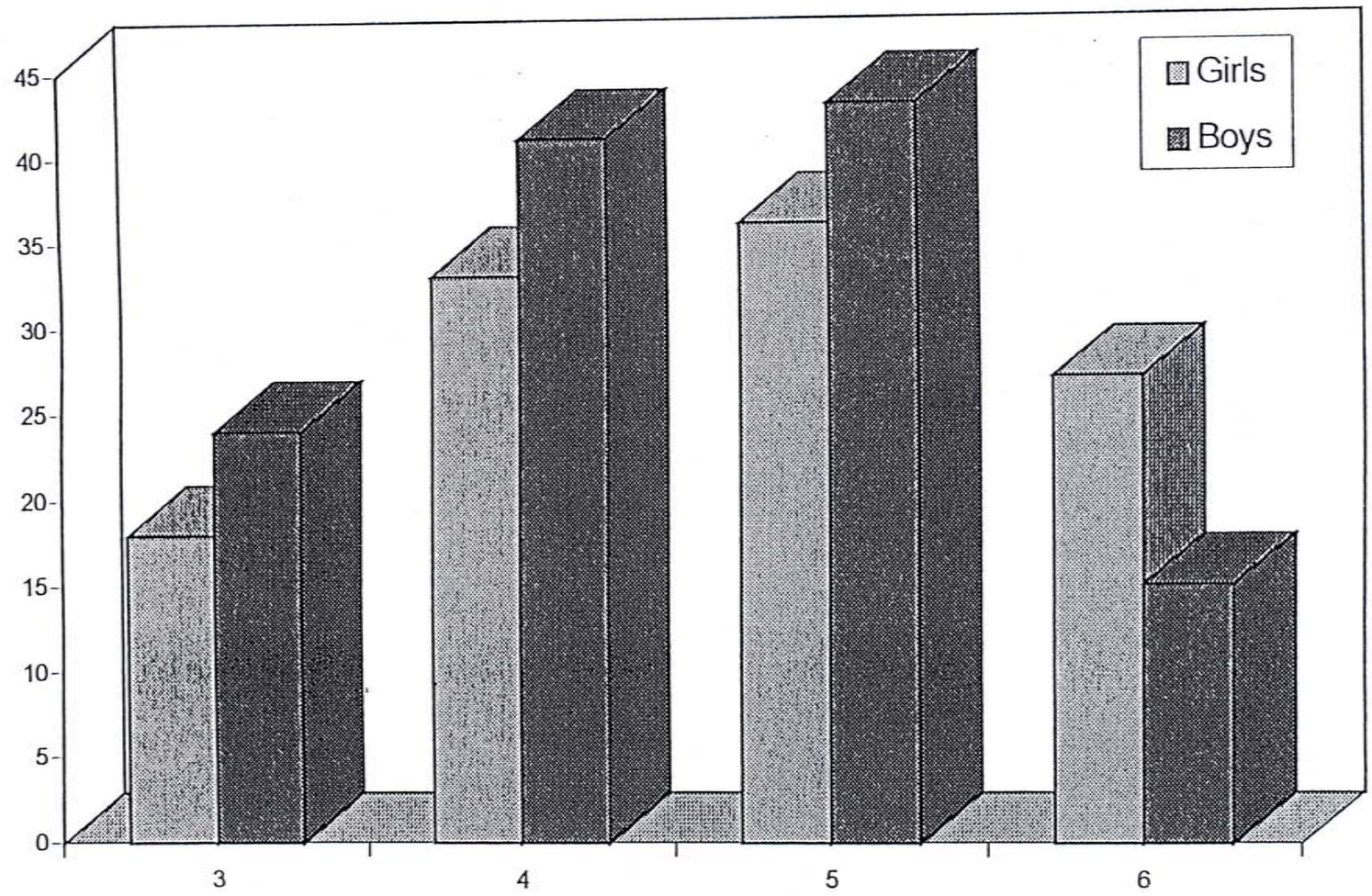


Figure 2. Distribution of Refraction

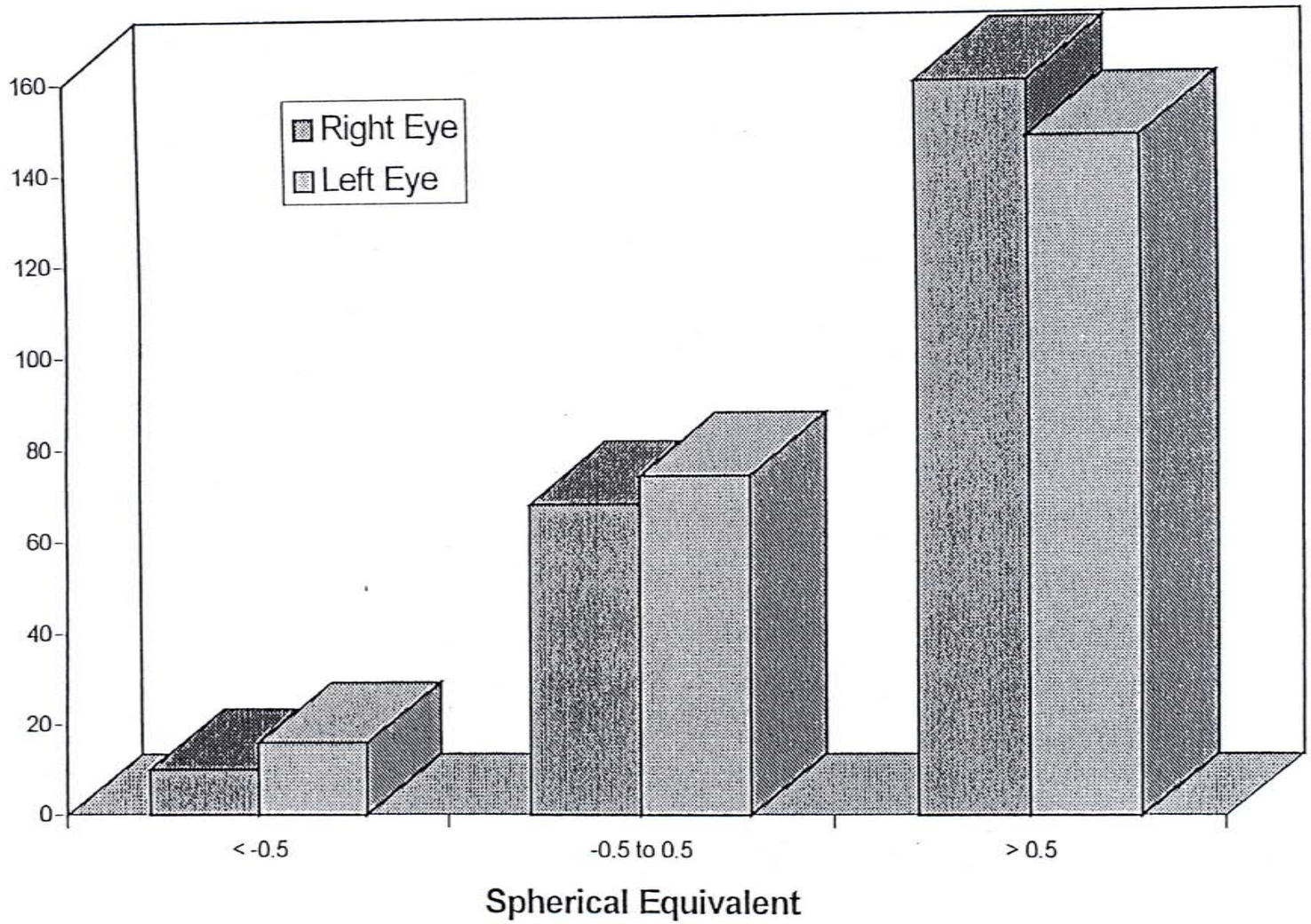


Figure 3. Distribution of Axial Length

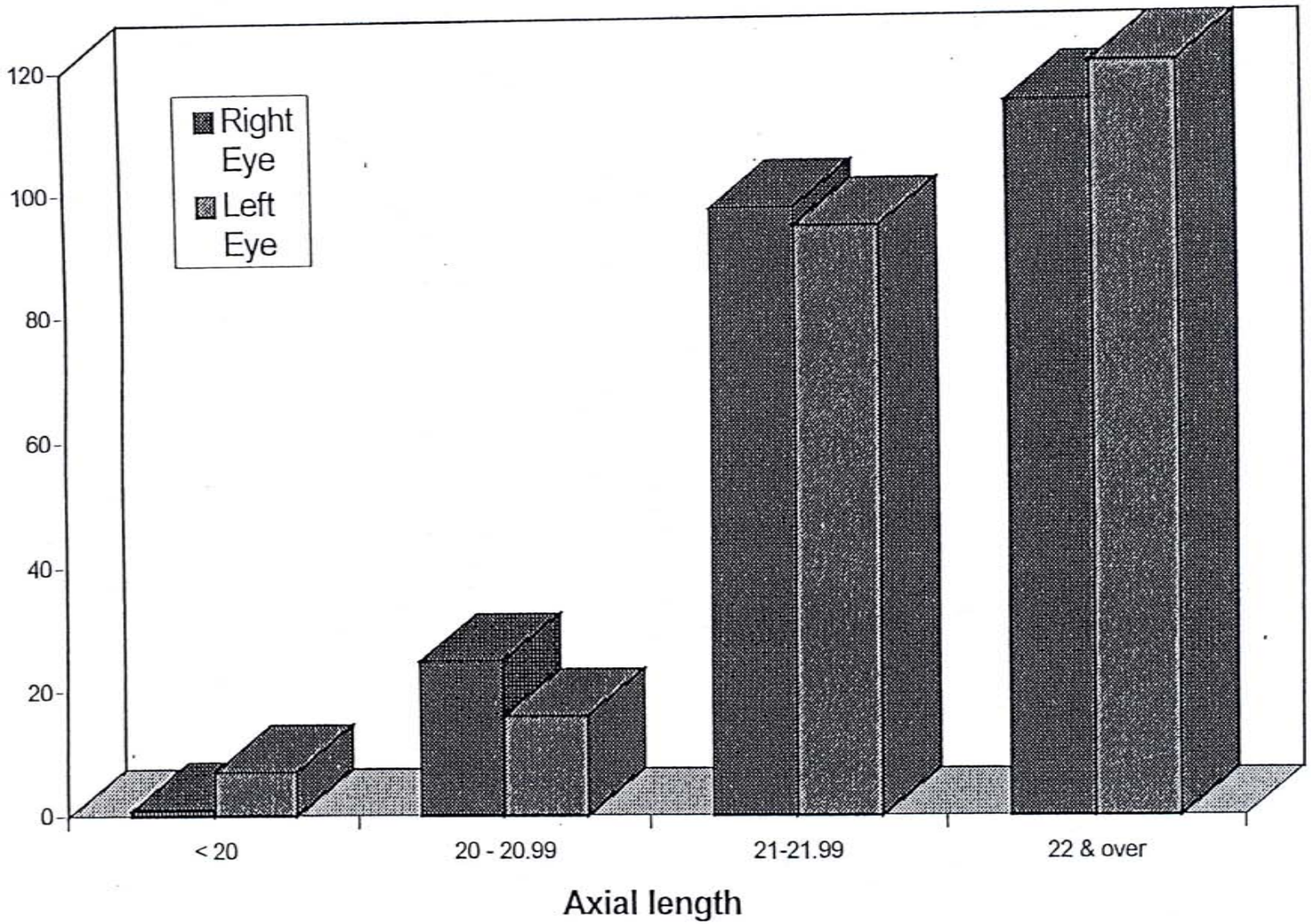


Figure 4. Distribution of Vitreous Length

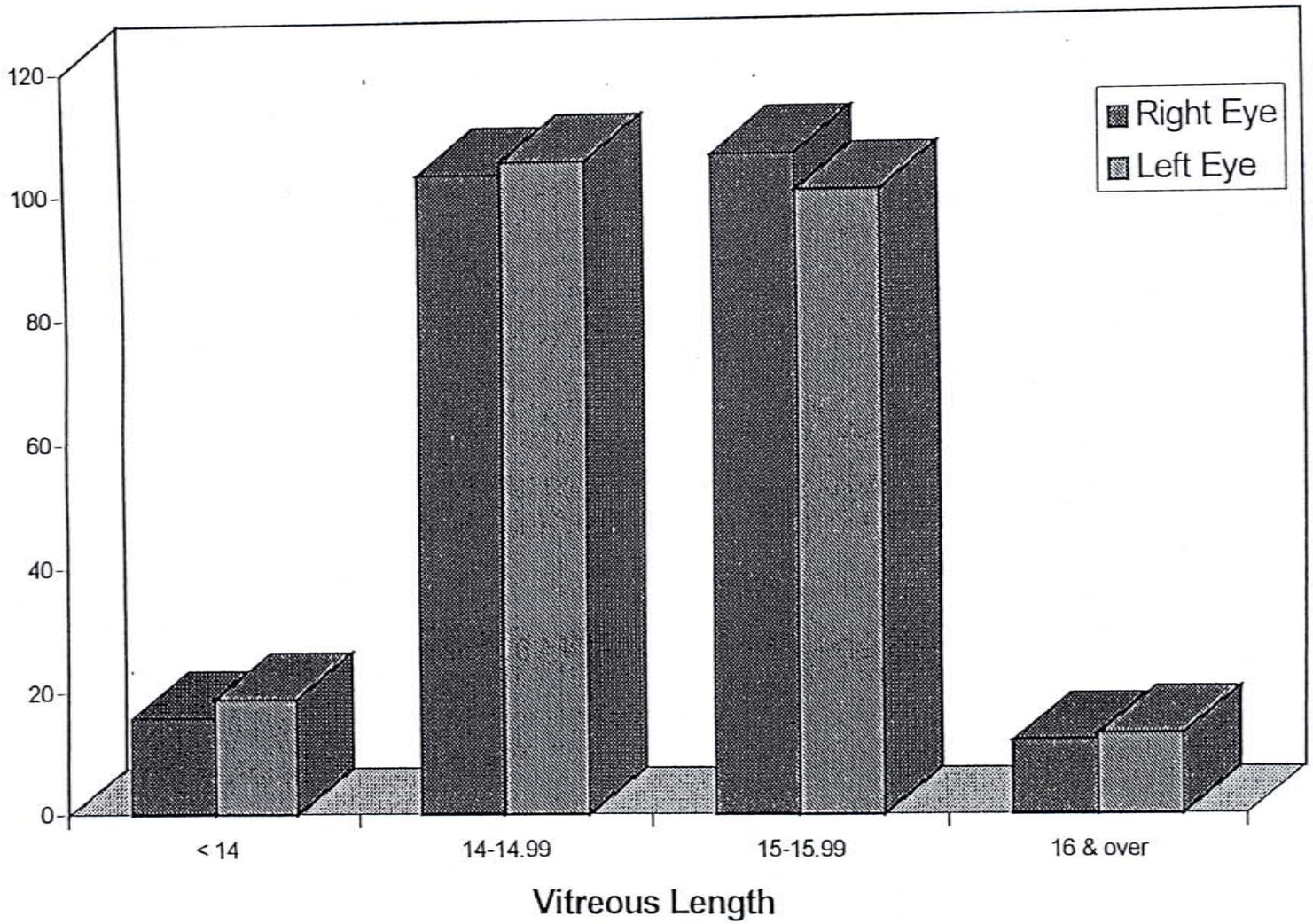


Figure 5. Scatterplot of Axial Length vs Age

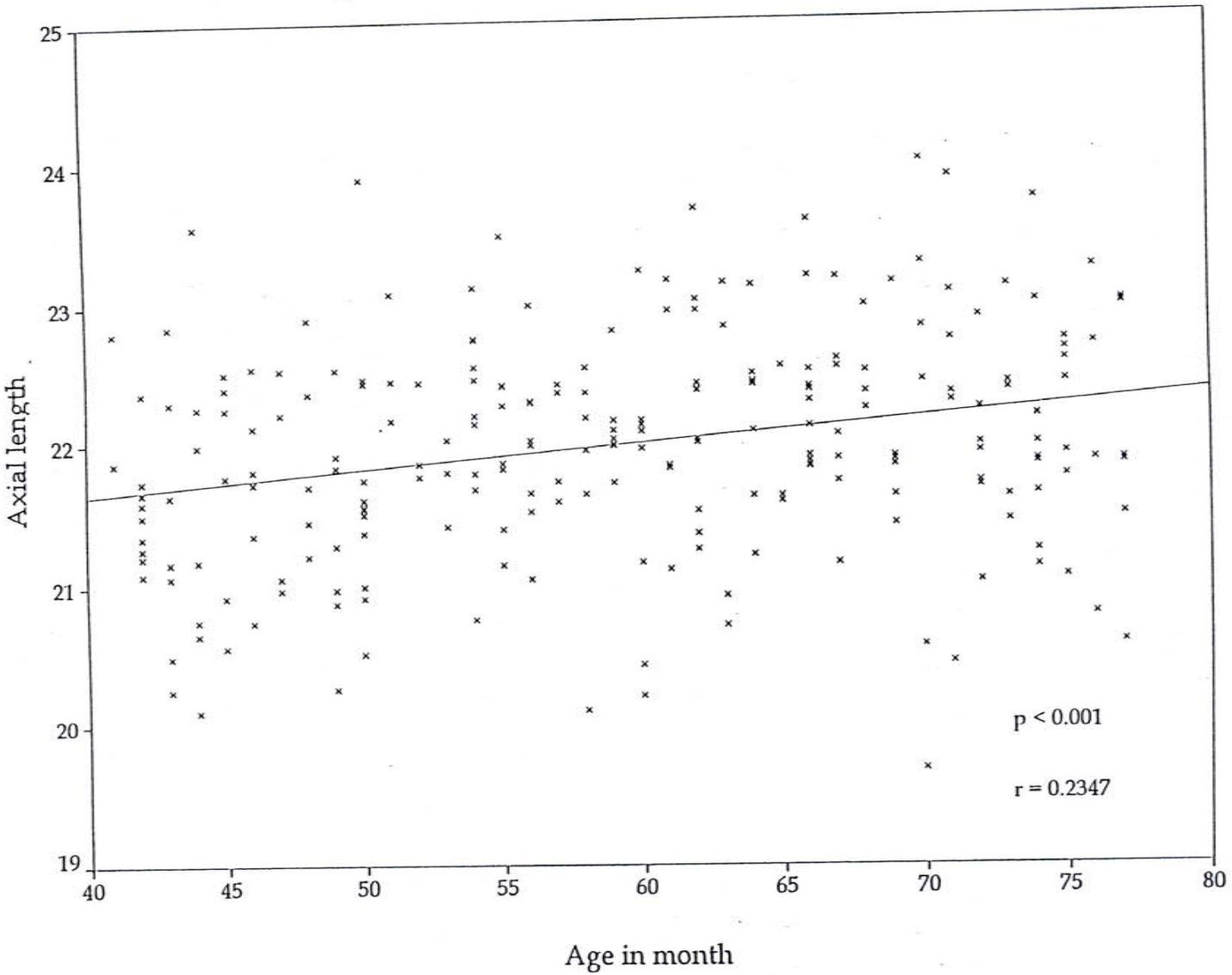


Figure 6. Scatterplot of Vitreous Length vs Age

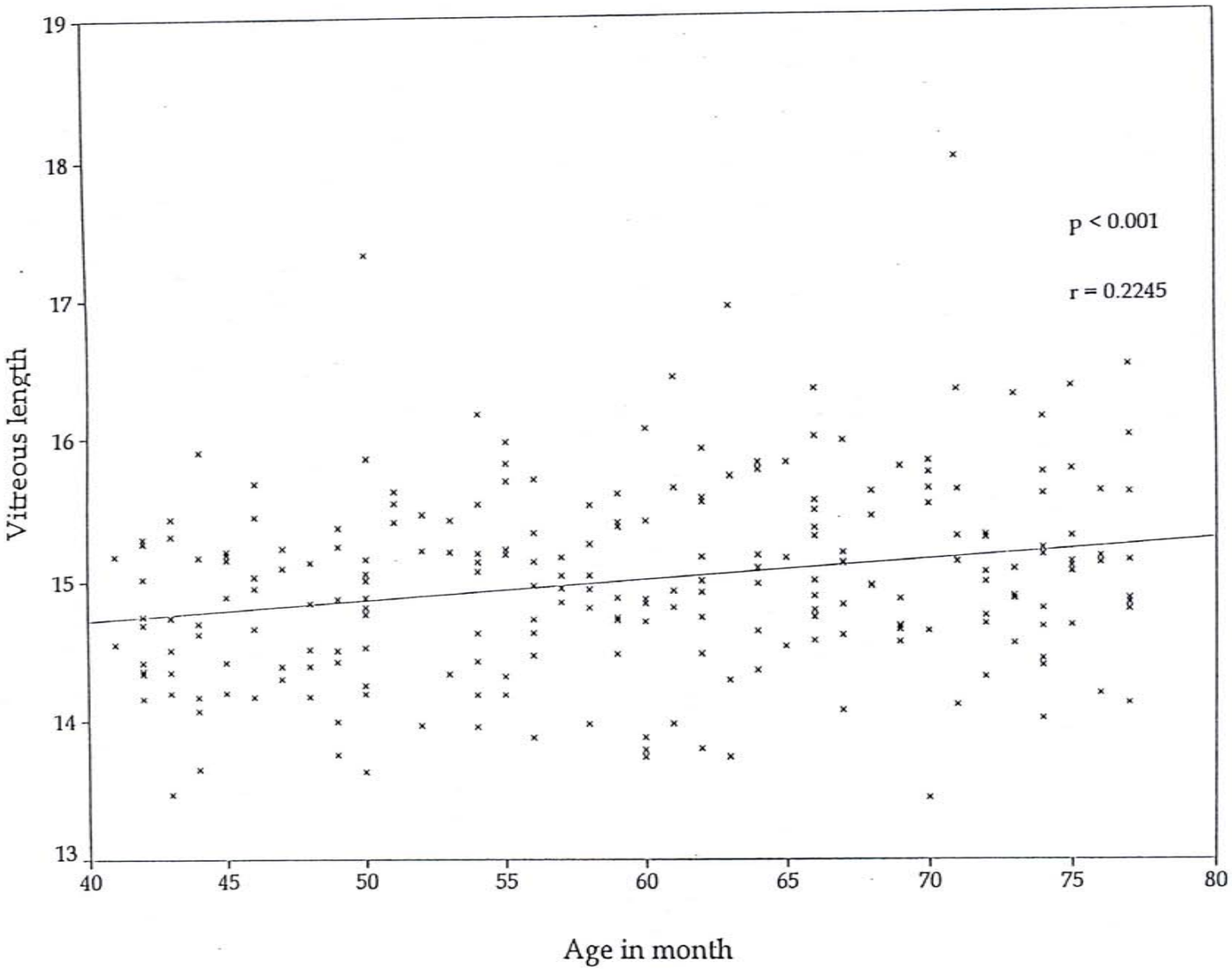


Figure 7. Scatterplot of Refraction vs Axial Length

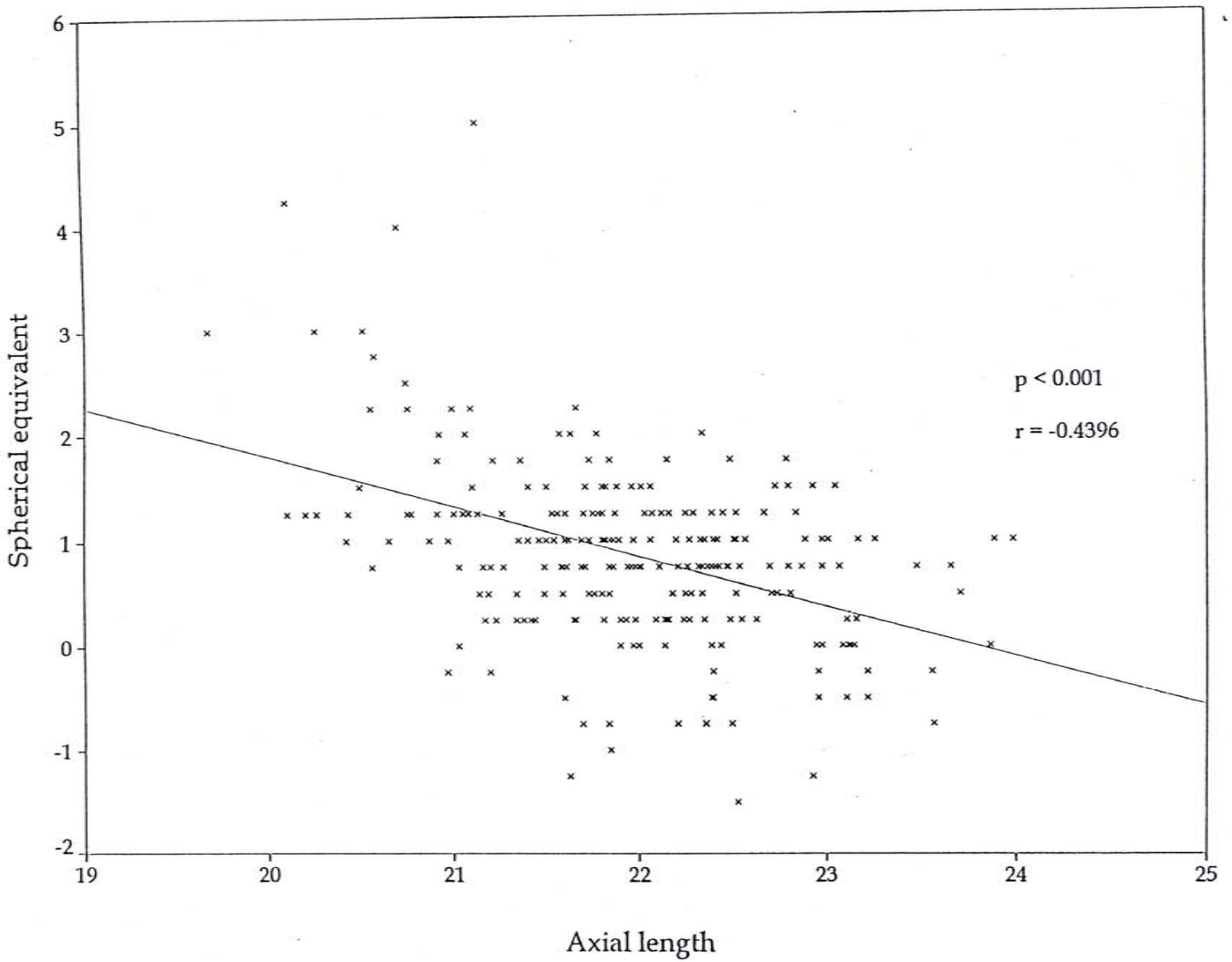


Figure 8. Scatterplot of Refraction vs Vitreous Length

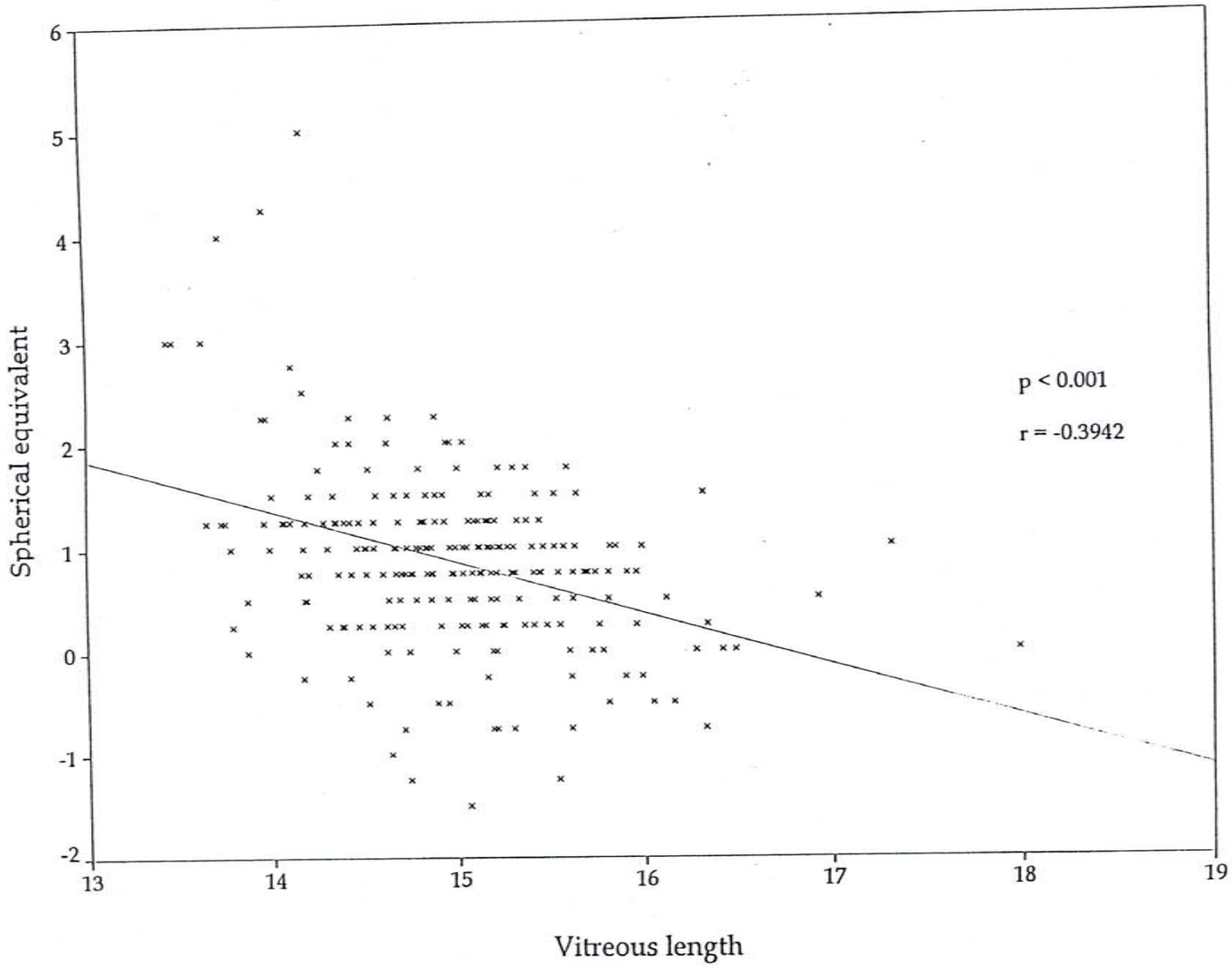


Figure 9: 95% C.I. for Axial Length

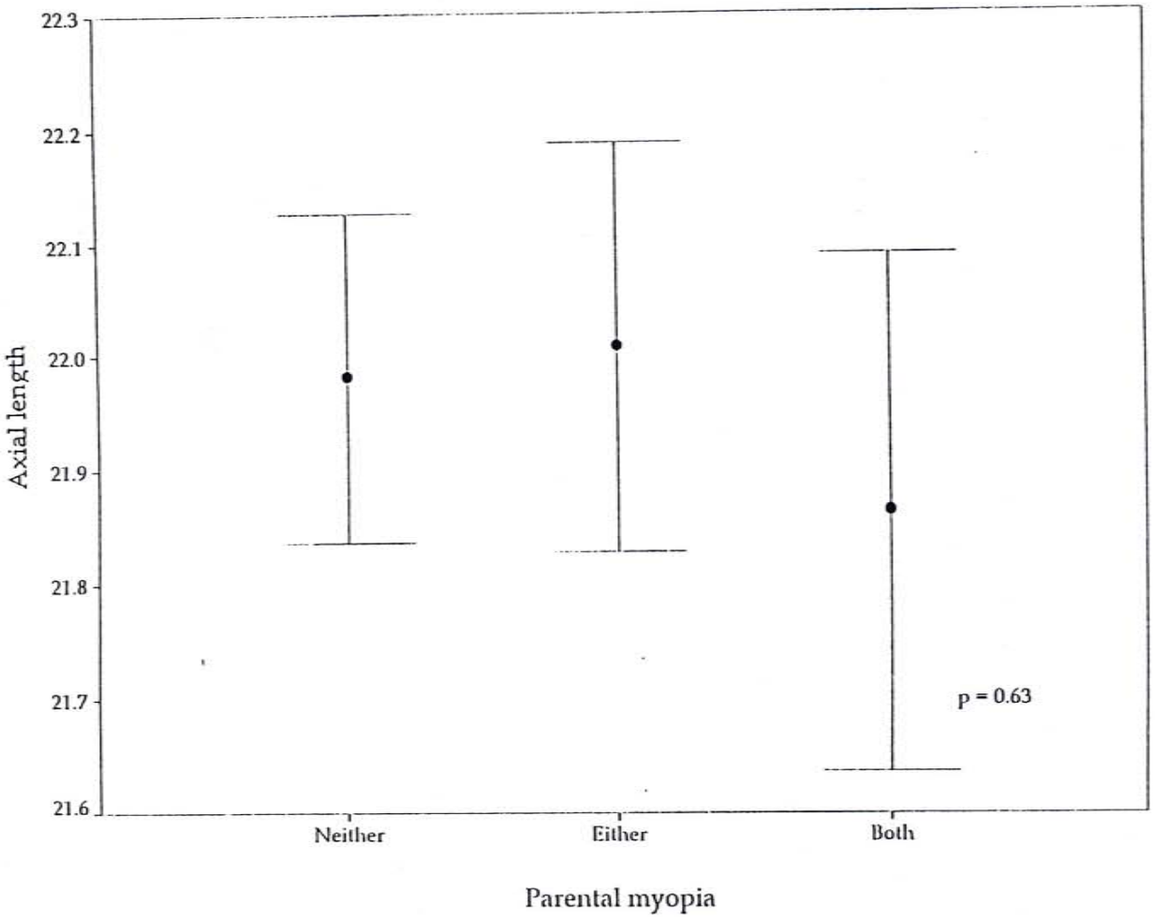


Figure 10: 95% C.I. for Vitreous Length

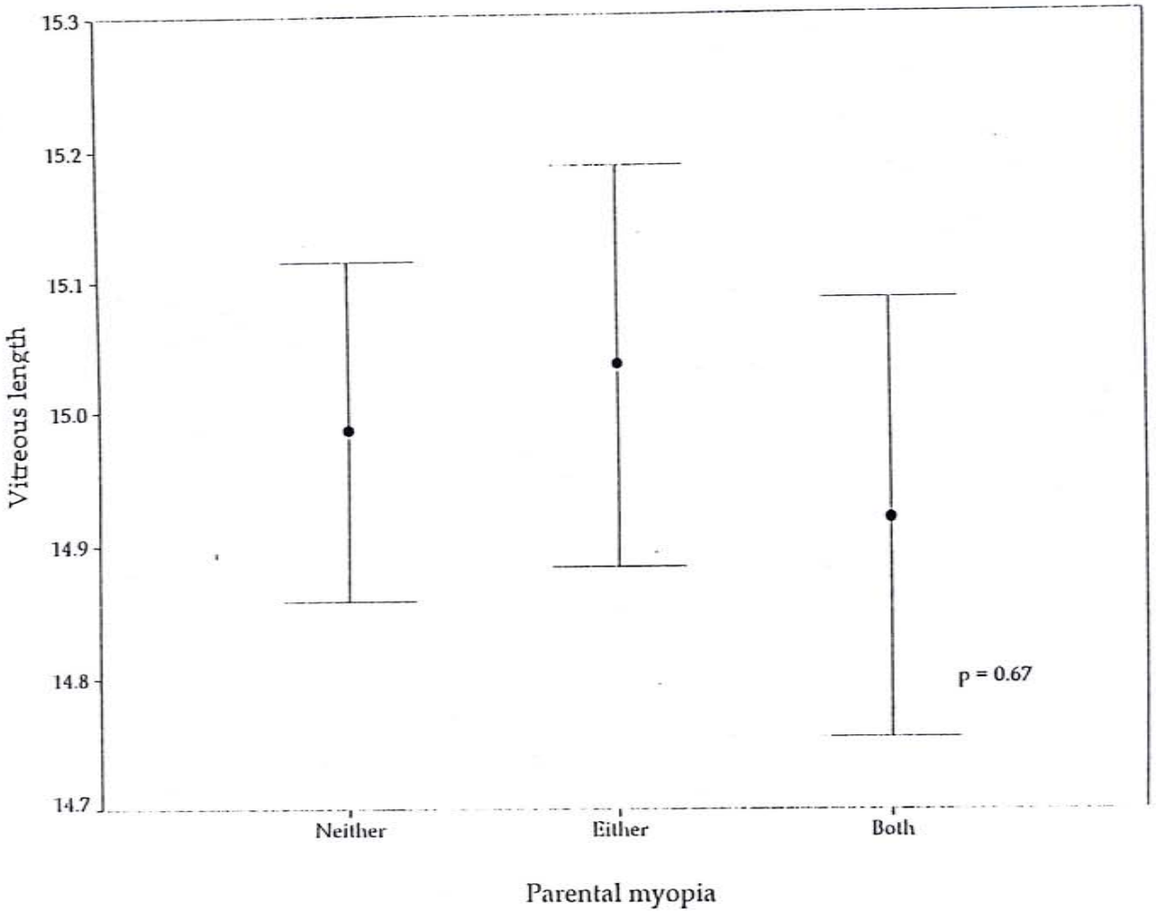


Figure 11: 95% C.I. for Spherical Equivalent

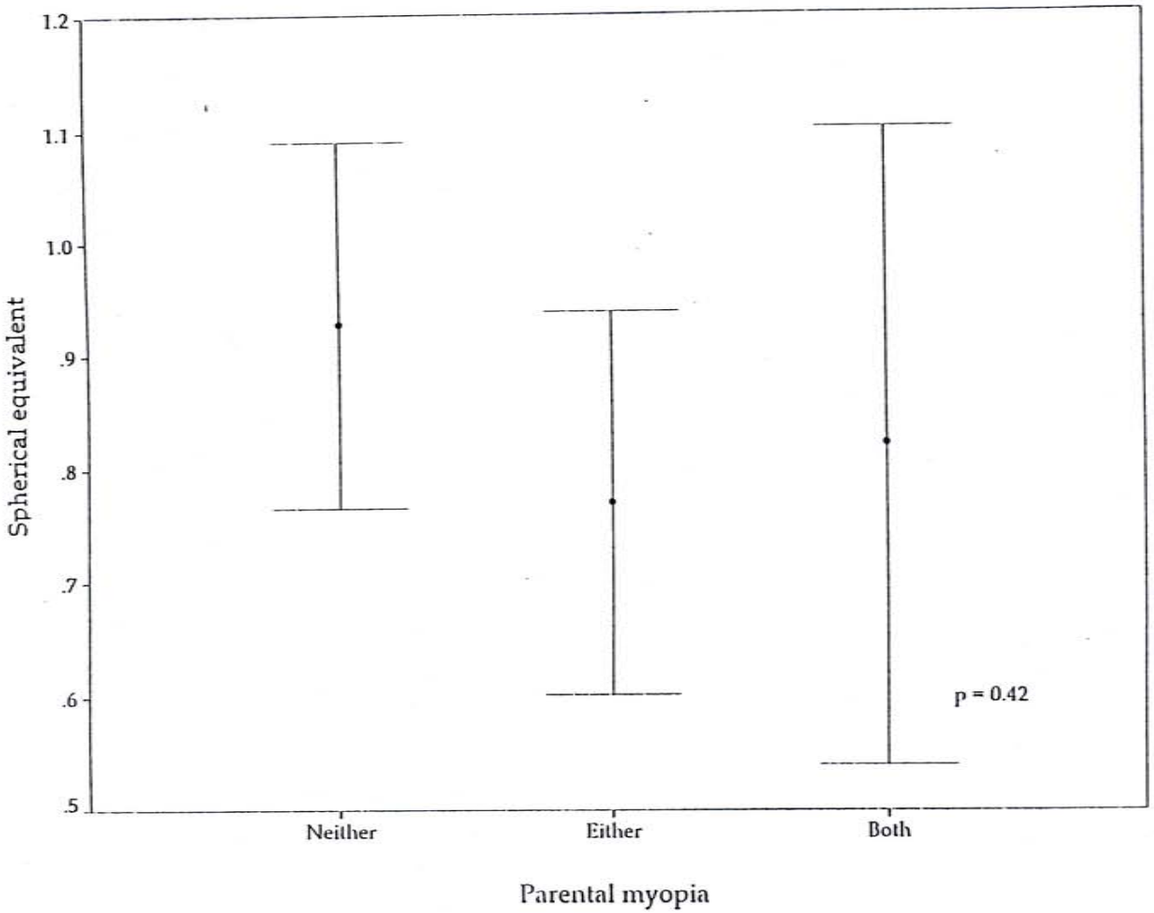


Figure 12: 95% C.I. for Spherical Equivalent

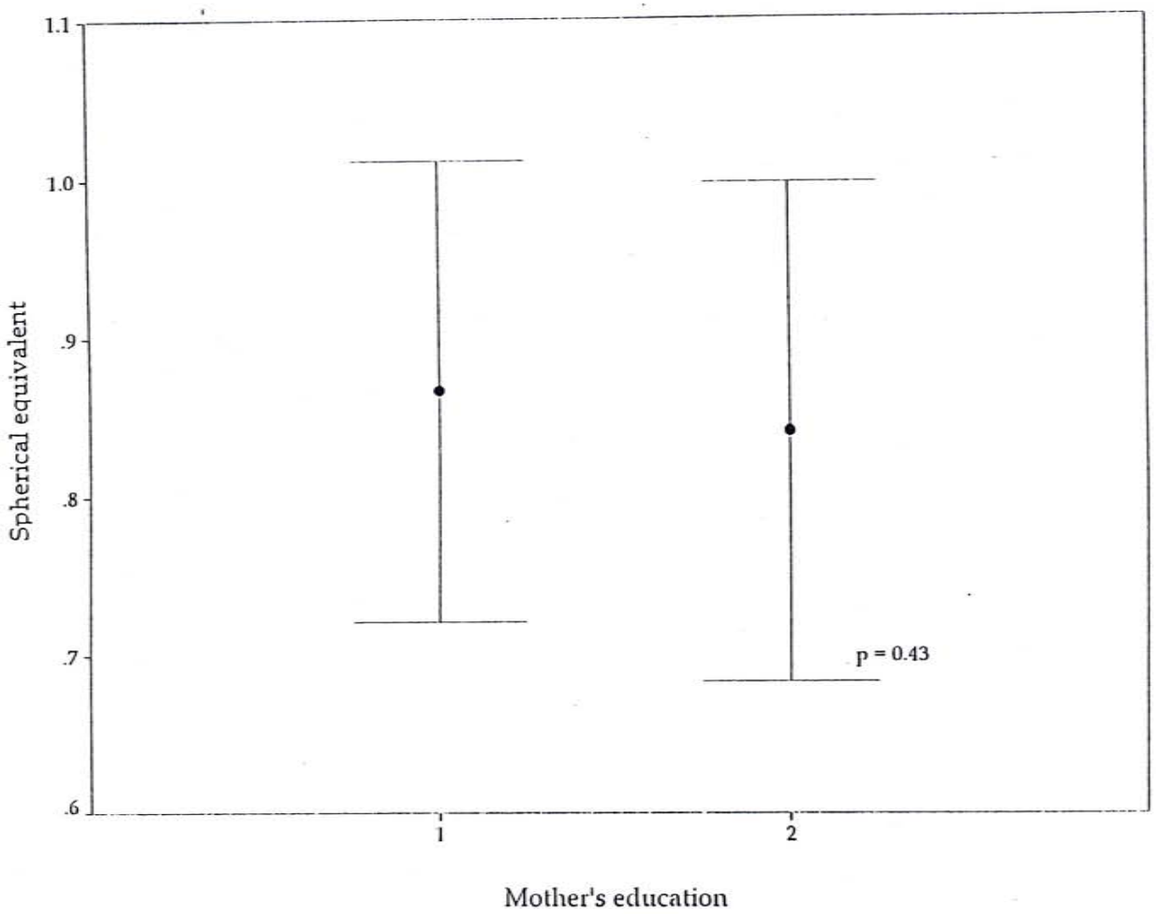
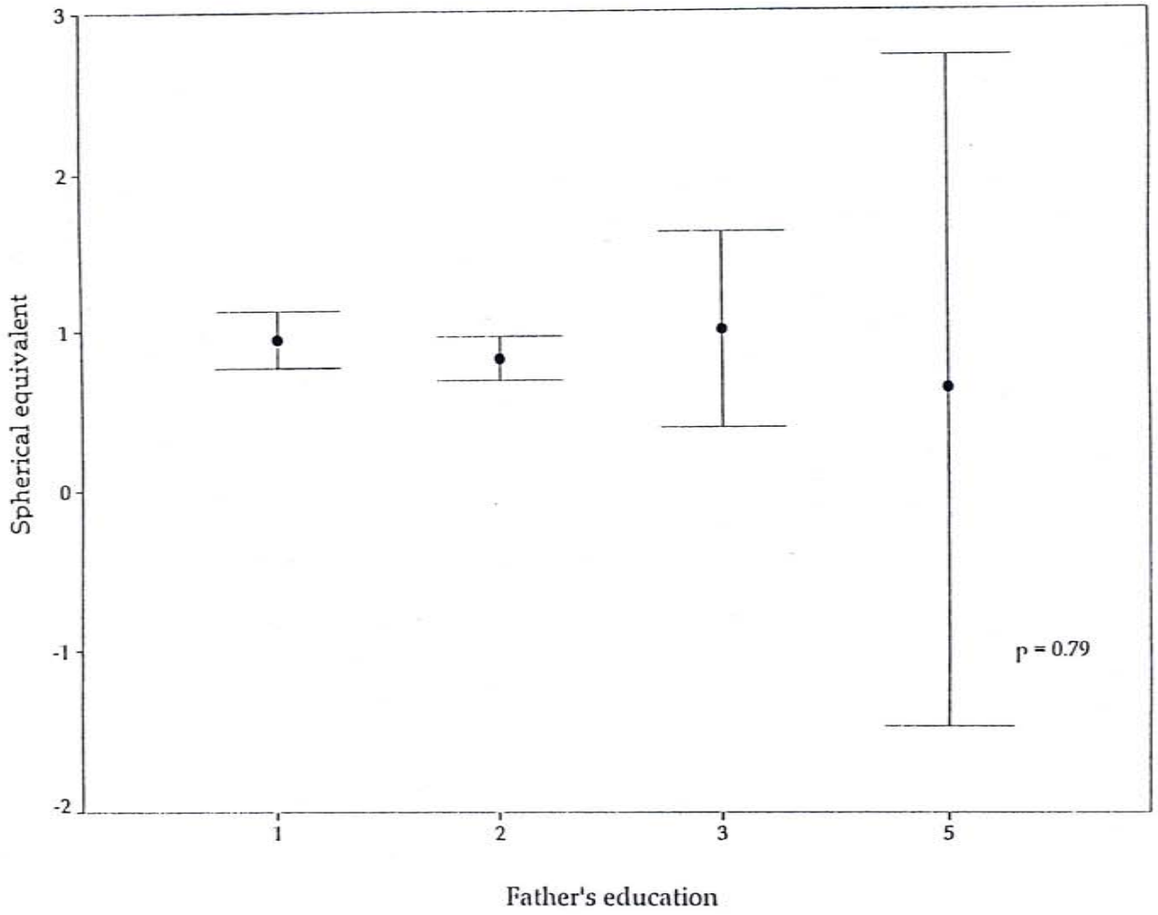


Figure 13: 95% C.I. for Spherical Equivalent



APPENDIX I-II

QUESTIONNAIRE IN CHINESE

APPENDIX I

學前兒童眼睛護理(非本校學生專用)

姓名:_____(　　)編號:_____

性別: 男/女　年齡:____ 出生日期:____年____月____日 出世紙號碼:_____

主要疾病:　手術 _____

其它 _____

眼睛病歷:　手術 _____

其它 _____

父母:

母親姓名:_____(　　) 年齡:____ 出生日期:____年____月____日

教育程度:　小學畢業或以下____　中學畢業____　預科畢業____
大專畢業____　大學畢業或以上____

有否近視　有/沒有

開始配帶近視眼鏡年齡　_____

父母親及兄弟姐妹是否有深近視（六百度或以上）　有/沒有

父親姓名:_____(　　) 年齡:____ 出生日期:____年____月____日

教育程度:　小學畢業或以下____　中學畢業____　預科畢業____
大專畢業____　大學畢業或以上____

有否近視　有/沒有

開始配帶近視眼鏡年齡　_____

父母親及兄弟姐妹是否有深近視（六百度或以上）　有/沒有

地址:_____

_____

電話:（家）_____（辦）_____（聯絡人）_____

家居日誌　　姓名 :_____(　　)編号:_____

書寫　　:　距離:_____厘米
閱讀　　:　距離:_____厘米
電視　　:　尺寸:_____厘米　距離:_____厘米
電腦　　:　尺寸:_____厘米　距離:_____厘米
電視遊戲機 :螢幕尺寸:_____厘米　距離:_____厘米

圖畫及拼砌遊戲等　　:　距離:_____厘米
電子遊戲機 (Gameboy)　:　距離:_____厘米

7·5·1995
(星期日)

	上午										下午													
	七時		八時		九時		十時		十一時		十二時		一時		二時		三時		四時		五時		六時	
書寫																								
閱讀																								
圖畫及拼砌遊戲等																								
電子遊戲機																								
電視																								
電腦																								
電視遊戲機																								

	下午																							
	七時		八時		九時		十時		十一時		十二時		一時		二時		三時		四時		五時		六時	
書寫																								
閱讀																								
圖畫及拼砌遊戲等																								
電子遊戲機																								
電視																								
電腦																								
電視遊戲機																								

撮要(確實用眼時間的總和):
書寫 :_____分鐘　閱讀 :_____分鐘　電子遊戲機 :_____分鐘　圖畫及拼砌遊戲等 :_____分鐘
電視 :_____分鐘　電腦 :_____分鐘　電視遊戲機 :_____分鐘

其它 :_____

8·5·1995 (星期一)

上午 下午

書寫
閱讀
圖畫及拼砌遊戲等
電子遊戲機
電視
電腦
電視遊戲機

撮要(確實用眼時間的總和):

書寫 :_____分鐘 閱讀 :_____分鐘 電子遊戲機 :_____分鐘 圖畫及拼砌遊戲等 :_____分鐘

電視 :_____分鐘 電腦 :_____分鐘 電視遊戲機 :_____分鐘

其它 :_____

9·5·1995 (星期二)

上午 下午

書寫
閱讀
圖畫及拼砌遊戲等
電子遊戲機
電視
電腦
電視遊戲機

撮要(確實用眼時間的總和):

書寫 :_____分鐘 閱讀 :_____分鐘 電子遊戲機 :_____分鐘 圖畫及拼砌遊戲等 :_____分鐘

電視 :_____分鐘 電腦 :_____分鐘 電視遊戲機 :_____分鐘

其它 :_____

10·5·1995 (星期三)

撮要(確實用眼時間的總和):

書寫 :_____分鐘　閱讀 :_____分鐘　電子遊戲機 :_____分鐘　圖畫及拼砌遊戲等 :_____分鐘

電視 :_____分鐘　電腦 :_____分鐘　電視遊戲機 :_____分鐘

其它 :_____

11·5·1995 (星期四)

撮要(確實用眼時間的總和):

書寫 :_____分鐘　閱讀 :_____分鐘　電子遊戲機 :_____分鐘　圖畫及拼砌遊戲等 :_____分鐘

電視 :_____分鐘　電腦 :_____分鐘　電視遊戲機 :_____分鐘

其它 :_____

12·5·1995(星期五)

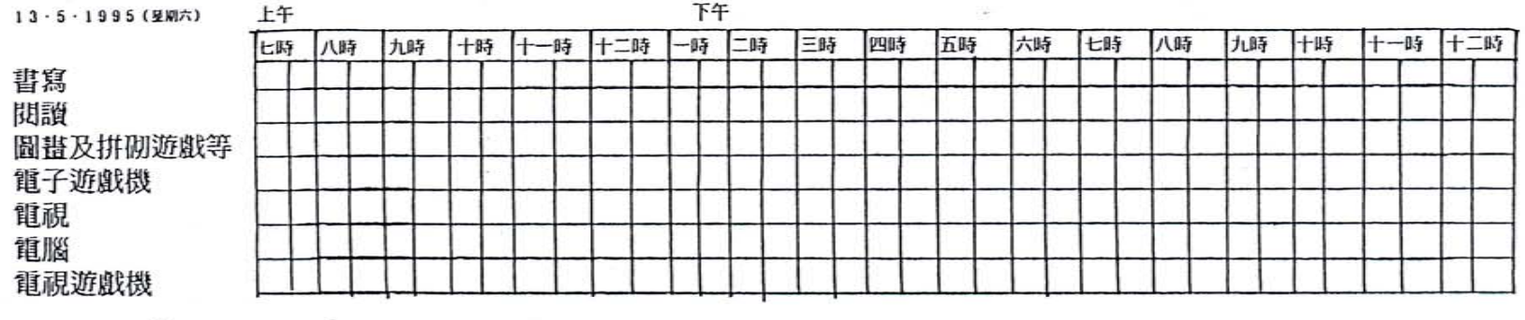
撮要(確實用眼時間的總和):

書寫 :_____分鐘 閱讀 :_____分鐘 電子遊戲機 :_____分鐘 圖畫及拼砌遊戲等 :_____分鐘

電視 :_____分鐘 電腦 :_____分鐘 電視遊戲機 :_____分鐘

其它 :_____

13·5·1995(星期六)



撮要(確實用眼時間的總和):

書寫 :_____分鐘 閱讀 :_____分鐘 電子遊戲機 :_____分鐘 圖畫及拼砌遊戲等 :_____分鐘

電視 :_____分鐘 電腦 :_____分鐘 電視遊戲機 :_____分鐘

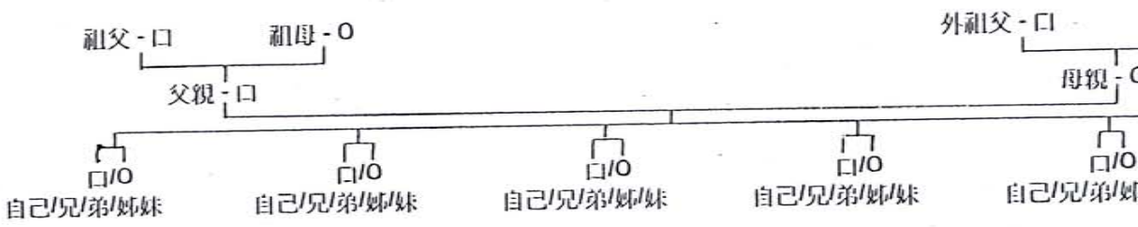
其它 :_____

檔案編號：_____

合家同歡齊驗眼(家庭成員表)

參加者請在□或○加上X符號

□: 男性　○:女性



身份	姓名	性別/歲數(出生日期)	身份証號碼	已知的主要眼疾/眼手術	其他主要疾病
自己	_____(　　)	男/女____(　　)	_____	_____	_____
祖父	_____(　　)	男/女____(　　)	_____	_____	_____
祖母	_____(　　)	男/女____(　　)	_____	_____	_____
外祖父	_____(　　)	男/女____(　　)	_____	_____	_____
外祖母	_____(　　)	男/女____(　　)	_____	_____	_____
父親	_____(　　)	男/女____(　　)	_____	_____	_____
母親	_____(　　)	男/女____(　　)	_____	_____	_____
兄/弟/姊/妹	_____(　　)	男/女____(　　)	_____	_____	_____
兄/弟/姊/妹	_____(　　)	男/女____(　　)	_____	_____	_____
兄/弟/姊/妹	_____(　　)	男/女____(　　)	_____	_____	_____
兄/弟/姊/妹	_____(　　)	男/女____(　　)	_____	_____	_____

地址　：_____

電話　：(家居) _____　(辦公室) _____　(聯絡人) _____

APPENDIX II

學校日誌

姓名 : _____( )

編号 : _____

書寫 : 距離：_____厘米

圖畫及拼砌遊戲等 : 距離：_____厘米

閱讀 : 距離：_____厘米

電視 : 尺寸：_____厘米 距離：_____厘米

電腦 : 尺寸：_____厘米 距離：_____厘米

8 · 5 · 1995（星期一）

	上午						下午					
	九時		十時		十一時		一時		二時		三時	
書寫												
閱讀												
圖畫及拼砌遊戲等												
電視												
電腦												

撮要(確實用眼時間的總和)：

書寫 ：_____分鐘

閱讀 ：_____分鐘

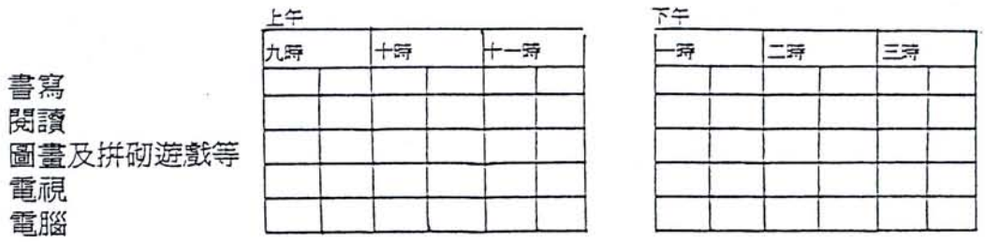
圖畫及拼砌遊戲等 ：_____分鐘

電視 ：_____分鐘

電腦 ：_____分鐘

其它 ：_____

9 · 5 · 1995（星期二）

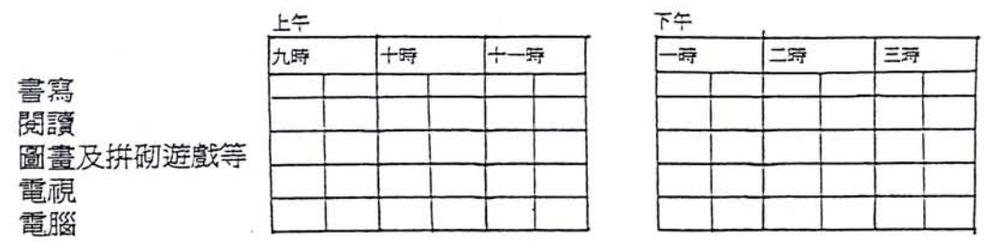


撮要(確實用眼時間的總和)：

書寫 ：_____分鐘
閱讀 ：_____分鐘
圖畫及拼砌遊戲等 ：_____分鐘
電視 ：_____分鐘
電腦 ：_____分鐘

其它 ：_____

10 · 5 · 1995（星期三）

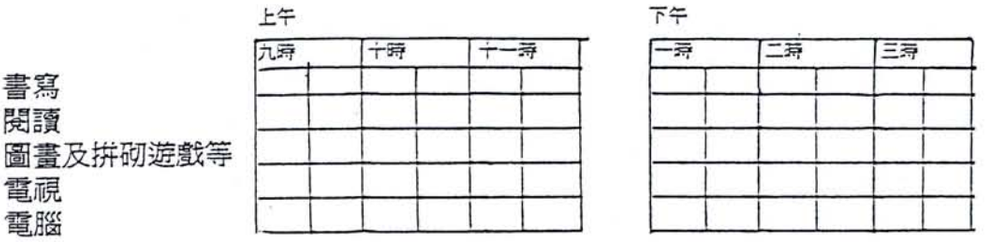


撮要(確實用眼時間的總和)：

書寫 ：_____分鐘
閱讀 ：_____分鐘
圖畫及拼砌遊戲等 ：_____分鐘
電視 ：_____分鐘
電腦 ：_____分鐘

其它 ：_____

11 · 5 · 1995（星期四）

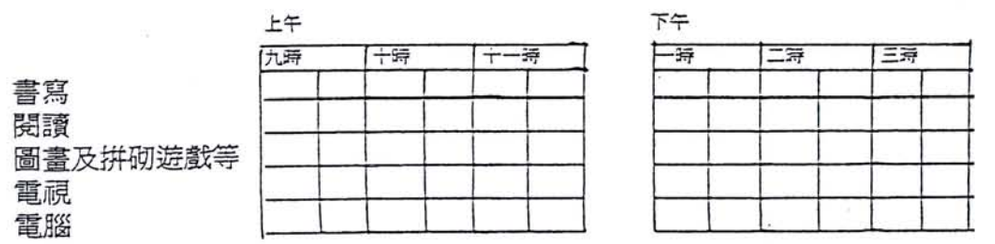


撮要(確實用眼時間的總和)：

書寫 ：_____分鐘
閱讀 ：_____分鐘
圖畫及拼砌遊戲等 ：_____分鐘
電視 ：_____分鐘
電腦 ：_____分鐘

其它 ：_____

12 · 5 · 1995（星期五）



撮要(確實用眼時間的總和)：

書寫 ：_____分鐘
閱讀 ：_____分鐘
圖畫及拼砌遊戲等 ：_____分鐘
電視 ：_____分鐘
電腦 ：_____分鐘

其它 ：_____

APPENDIX III-IV

QUESTIONNAIRE IN ENGLISH TRANSLATION

APPENDIX III

Data Sheet (Children): Name:_____ Serial No:_____

Before Pupil Dilation:

VA: (OD) _____ PH _____ Pupils: NAD: _____APD: _____

(OS) _____ PH _____ Others:_____

Eye Examinations: # before and after pupil dilatation

NAD: _____
Squint: _____
Amblyopia: _____
Allergic Conj.:_____
**Cataract: _____
Glaucoma: _____
Others:_____

*Auto-K: (OD) _____

(OS) _____

*Auto-R: (OD) _____

(OS) _____

*NCT: (OD) _____ _____ _____ _____ _____mmHg Mean _____ mmHg

(OS) _____ _____ _____ _____ _____mmHg Mean _____ mmHg

After Pupil Dilation:

*Auto-K: (OD) _____

(OS) _____

*Auto-R: (OD) _____

(OS) _____

*NCT: (OD) _____ _____ _____ _____ _____mmHg Mean _____ mmHg

(OS) _____ _____ _____ _____ _____mmHg Mean _____ mmHg

*Axial Length: (OD) _____ _____ _____ _____ _____mm Mean _____ mm

(OS) _____ _____ _____ _____ _____mm Mean _____ mm

*Three (3) readings are enough if they are consistent

Data Sheet (Adult): Name:_____ Serial No:_____

Before Pupil Dilation:

VA: (OD) _____ PH _____ Pupils: NAD: _____APD: _____

(OS) _____ PH _____ Others:_____

Eye Examinations:(before and after pupil dilatation)

NAD: _____
Squint: _____
Amblyopia: _____
Allergic Conj.:_____
Cataract: _____
Glaucoma: _____
ARM: _____DMR: _____HTR: _____Others:_____

*Auto-K: (OD) _____

(OS) _____

*Auto-R: (OD) _____

(OS) _____

*NCT: (OD) _____ _____ _____ _____ _____mmHg Mean _____ mmHg

(OS) _____ _____ _____ _____ _____mmHg Mean _____ mmHg

After Pupil Dilation:

*Auto-K: (OD) _____

(OS) _____

*Auto-R: (OD) _____

(OS) _____

*NCT: (OD) _____ _____ _____ _____ _____mmHg Mean _____ mmHg

(OS) _____ _____ _____ _____ _____mmHg Mean _____ mmHg

*Axial Length: (OD) _____ _____ _____ _____ _____mm Mean _____ mm

(OS) _____ _____ _____ _____ _____mm Mean _____ mm

*Three (3) readings are enough if they are consistent

APPENDIX IV

Eye Care For Pre-school Children

Name: _____( ) Serial No:_____

Sex / Age (DOB):_____( ) ID No: _____

Major illness: Operation:_____

Others:_____

Ocular history: Operation:_____

Others:_____

Parents:

Mother: Name_____( )Age (DOB):_____

Education Level: Up to primary school graduate _____
Secondary school graduate _____
Matriculation _____
College _____
University graduate or above _____

Do you have myopia: _____

Age start wearing myopia glasses: _____

Family history of high myopia (6 D or more): _____

Father: Name_____( )Age (DOB):_____

Education Level: Up to primary school graduate _____
Secondary school graduate _____
Matriculation _____
College graduate _____
University graduate or above _____

Do you have myopia: _____

Age start wearing myopia glasses: _____

Family history of high myopia (6 D or more): _____

Address:_____

_____

Tel:_____(Home)_____(Office: contact)

DIARY AT SCHOOL

Writing:	Working distance:	_____cm			
Reading:	Working distance:	_____cm			
Gameboy:	Playing distance:	_____cm			
Television:	Size:	_____cm	Watching distance:	_____cm	
Computor:	Size:	_____cm	Working distance:	_____cm	
Video-game:	Size of monitor:	_____cm	Playing distance:	_____cm	

7/5/95 (Sunday):

	8am	9am	10am	11am	12am	1pm	2pm	3pm	4pm
Writing:									
Reading:									
Computor:									
TV:									
Videogame:									
Gameboy:									

7/5/95 (Sunday): SUMMARY

Writing:	_____Hrs	Reading:	_____Hrs	Computor:	_____Hrs
Watching TV:	_____Hrs	Video-game:	_____Hrs	Gameboy:	_____Hrs

Other remarks: _____

DIARY AT HOME

Writing:	Working distance:	_____cm		
Reading:	Working distance:	_____cm		
Gameboy:	Playing distance:	_____cm		
Television:	Size:	_____cm	Watching distance:	_____cm
Computor:	Size:	_____cm	Working distance:	_____cm
Video-game:	Size of monitor:	_____cm	Playing distance:	_____cm

7/5/95 (Sunday):

	7am	8am	9am	10am	11am	12am	1pm	2pm	3pm	4pm	5pm	6pm	7pm	8pm	9pm	10pm	11pm
Writing:																	
Reading:																	
Computor:																	
TV:																	
Videogame:																	
Gameboy:																	

7/5/95 (Sunday): SUMMARY

Writing:	_____Hrs	Reading:	_____Hrs	Computor:	_____Hrs
Watching TV:	_____Hrs	Video-game:	_____Hrs	Gameboy:	_____Hrs

Other remarks: _____

CONSENT

I _____ (name) hereby consent on my son/daughter _____'s (name of the child) behalf to undergo the " Pre-schoolers Eye Screening and Causes of Myopia Study".

This will involve a series of questionnaires, and examination of his/her eyes which are routinely done in many eye clinics. The tests are in no way harmful to his/her eyes. Eyedrops will be used to ensure accurate results during the examination. They are proved to be safe to use in young children without any drastic side-effect. The purpose, benefits, potential risks and complications of the study have been explained to me by Dr _____/_____.

_____ _____
Name in block letter Signature

_____ _____
Name of witness Signature

_____ _____
Name of doctor/staff Signature

Date:_____

眼睛檢查同意書

一九九____年____月____日
本人_____同意小兒/小女_____接受"幼兒護眼及近視成因"研究計劃。
此項研究計劃主要替孩子進行一般性的眼睛檢查，故無害於兒童眼睛的健康。爲提高測試效果的準確程度，於檢查時，醫護人員或工作人員將採用眼藥水進行有關檢查。此外，家長亦需要填寫有關問卷，以協助此項調查計劃。有關研究性質、效果及可能引起之危險和併發症已由_____醫生/有關工作人員向本人解釋清楚。
:_____

簽署:_____